Royal Commission on Matters of Health
and Safety Arising from the Use of
Asbestos in Ontario

Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980

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PROCEEDINGS OF SECOND PUBLIC MEETING

FRIDAY, DECEMBER 12, 1980

ONTARIO INSTITUTE FOR STUDIES IN EDUCATION

AUDITORIUM

9:30 a.m. - 4:00 p.m.

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A. Introduction

In April, 1980, Ontario's Minister of Labour, the Honourable Robert G. Elgie, M.D., announced in the Legislature the creation of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. The Commission's terms of reference are as follows:

(1) To investigate all matters relating to health and safety arising from the use of asbestos in Ontario;

(2) To identify the relevant data related to asbestosis, mesothelioma, and other diseases and health hazards of persons working with, or exposed to, asbestos in Ontario;

(3) To review the present basis for Workmen's Compensation Board awards as they relate to occupational health matters affecting workers exposed to asbestos, including any special programmes dealing with the rehabilitation of such workers;

(4) To make such recommendations in relation to the above as the Commission deems appropriate.

The Minister of Labour named Dr. J. Stefan Dupré to serve as Chairman of the Commission, and Dr. J. Fraser Mustard and Dr. Robert J. Uffen as Commissioners.

From a reading of the Commission's terms of reference, it is evident that the Royal Commission on Asbestos is concerned with questions which are often highly technical and complex. In a preliminary effort to explore some of these questions in an open forum, the Commission decided to hold two public meetings during the fall of 1980. These were meant to be educative sessions -- both for the Commission and the audience prior to the Commission's deadlines for written submissions and its schedule of public hearings. The Commission scheduled its meetings for Friday, October 31 and Friday, December 12, 1980.

Information regarding the public meetings was disseminated via a newspaper advertisement, a brochure, and a newsletter. As a result, approximately 230 people attended both the first and second public meetings.

What follows is a summary report of the proceedings of the Commission's second public meeting. A report of the proceedings of the first public meeting was published in December, 1980.

Royal Commission on Asbestos

Public Meeting #2

"Asbestos in the Context of General Issues"

Friday, December 12, 1980

Ontario Institute for Studies in Education Auditorium
252 Bloor Street West, Toronto

A G E N D A

9:00 a.m. - 9:30 a.m. — Registration

9:30 a.m. - 9:45 a.m. — Introduction by Royal Commission on Asbestos
Chairman, Dr. J. Stefan Dupré

9:45 a.m. - 10:15 a.m. — Address by Dr. Eric Chatfield, Ontario Research Foundation, on The Problems of Asbestos Measurement

10:15 a.m. - 10:45 a.m. — Question Period

Session Chairman: Dr. Robert J. Uffen

10:45 a.m. - 11:00 a.m. — Coffee Break

11:00 a.m. - 12:00 noon — The Health Effects of Asbestos and Other Hazardous Substances: Assessing the Quality of Scientific Evidence

(a) Address by Dr. David Sackett, Professor, Clinical Epidemiology and Biostatistics, McMaster University

(b) Address by Dr. David Muir, Director, Occupational Health Program, McMaster University

Session Chairman: Dr. J. Fraser Mustard

12:00 noon - 12:45 p.m. — Question Period

12:45 p.m. - 2:00 p.m. — Lunch Break
(Note: Lunch is not being served by the Commission.)

2:00 p.m. - 3:00 p.m. — Frameworks for the Regulation of Hazardous Substances

(a) Address by Dr. Steven Kelman, Harvard Kennedy School of Government, on Other Jurisdictions

(b) Address by Dr. G. Bruce Doern, School of Public Administration, Carleton University, on the Canadian Context

Session Chairman: Dr. Donald N. Dewees

3:00 p.m. - 3:45 p.m. — Question Period

B. Address by Dr. Eric J. Chatfield, Head, Electron Optical Laboratory, Department of Applied Physics, Ontario Research Foundation, on "The Problems of Measurement of Asbestos"

In the first part of the day's agenda, Commissioner Dr. Robert J. Uffen introduced Dr. Eric Chatfield of the Ontario Research Foundation.

Dr. Chatfield received his M.A. in Physics, Chemistry, and Crystallography from Cantab; and his Ph.D. in Colloid Chemistry from the same institution. Dr. Chatfield's areas of expertise span the fields of electron microscopy, electron diffraction, electron probe microanalysis, particle and materials characterization, air and water sampling, mineral identification, asbestos measurement, radiation measurements, and image analysis. Dr. Chatfield is widely published in these areas.

Among Dr. Chatfield's many professional affiliations, he has served on the Secretariat of the International Standards Organization's Working Group on Environmental Measurement; he has been a member, for Canada, of the Commission of European Communities' Asbestos Co-operation Subcommittee; and he has served as a member of the Expert Committee on Asbestos Analysis of the Ontario Ministry of the Environment.

A copy of the full text of Dr. Chatfield's address appears as Appendix A to these Proceedings.

In his talk, Dr. Chatfield outlined some of the difficulties in measuring asbestos fibres; in differentiating various types of asbestos fibres; and in distinguishing between asbestos and non-asbestos fibres, particularly when the samples are small. He explained in considerable detail the techniques developed for measuring asbestos in the workplace and in the general environment, and discussed their limitations.

He first explained that asbestos is a term used to describe a number of minerals that have "the property that they can be separated into long silky fibres." (A fibre is usually arbitrarily defined as a particle with a length-to-width ratio of 3:1. It has been suggested that the ratio should be incresed to 10:1 in order to reliably distinguish between asbestos and elongated non-asbestos fibres in the electron microscope.)

There are three main varieties of asbestos in commercial use: crysotile, amosite and crocidolite. The first is the most abundant and most used. All asbestos fibres are composed of a variety of elements; in fact, variations in composition can occur within the same species and even "from one fibre to the next in material from the same deposit."

Since no single element is uniquely associated with these minerals, it is impossible to detect or identify small amounts of asbestos using simple chemical analysis methods, Chatfield said.

The situation is further complicated by the fact that non-asbestos particles can have similar compositions. Unlike these other particles, asbestos particles are flexible, thin and long. The difference between asbestos and non-asbestos particles of the same mineral is concerned with

a property known as crystal habit (the shape in which the mineral grows). With large samples, it is possible to distinguish the asbestos and non-asbestos versions of the same mineral, but the same is not true when a single small particle of the mineral is examined under a microscope.

Dr. Chatfield noted that a precise definition of asbestos is important for the workplace because "this definition controls whether the individuals working in such an operation should be considered to be asbestos workers or not." He pointed out, for example, that many mining operations that do not primarily produce asbestos "have minerals in the ore body which are indistinguishable from asbestos and are of unknown health hazard." In addition, problems can arise when measurements are made near a mining or processing operation; it is then necessary to distinguish between fibres from these operations and other elongated particles present already.

Another problem is determining what size of asbestos particle to measure. "No one has so far been able to tell us what we should measure." Medical studies suggest that fibres less than 0.2 micrometres in diameter are most significant in causing cancer.

Dr. Chatfield noted that most asbestos fibres found in the environment and the workplace are too small to be resolved by optical microscopes. Such measurements as are made have limitations: they include all particles (not just asbestos fibres) with length-to-width ratios exceeding 3:1, and they cannot resolve fibres less than 0.2 micrometres in diameter, no matter how long they are.

Dr. Chatfield then turned to the question of measuring airborne asbestos in the workplace. He said that in a workplace in which asbestos is being used, one can assume that most of the fibres found are likely to be of the variety of asbestos being used. Thus, a simple and inexpensive measuring technique involving membrane filters can be used. Dr. Chatfield then described this measurement technique in some detail; it involves collecting fibres on membrane filters and examining them in optical microscopes, using a technique known as phase contrast illumination.

There are some deficiencies in this approach, however. Large differences can occur in the measurements of identical asbestos samples by different laboratories, caused by microscope misalignment and by operator inexperience and fatigue. It has also been discovered that discrepancies can occur in counting split or clumped fibres. Unfortunately, these factors can cause low results to be reported.

Furthermore, the validity of the membrane filter-optical microscope technique for assessing exposure to asbestos is questionable. In many samples, less than 1% of the total number of fibres may be recorded and it is not known whether the fibres observed in the optical microscope are in fact those responsible for health effects. Also, the phase contrast technique counts all fibres in the samples, not just asbestos fibres.

Turning to the question of measuring asbestos in the general environment (as opposed to the workplace), Dr. Chatfield said that the optical microscope technique is totally unsuitable for environmental sampling. Asbestos fibres in the general environment are usually too small in diameter to be detected by optical microscopes. Also, since asbestos and non-asbestos fibres are not distinguished, the total fibre count (which could include many non-asbestos fibres) may bear little relationship to the actual amount of asbestos in the air.

Dr. Chatfield then described in detail other methods of environmental measurement involving the use of the transmission electron microscope (which has a much greater resolution than the optical microscope) and X-ray techniques. He said that early criticisms of the electron microscopy techniques have now been shown to be unjustified and good agreement within and between laboratories has been achieved.

In many cases, identifying asbestos fibres with these techniques is much more involved, and Dr. Chatfield outlined several complications. For example, the arbitrary definition of asbestos fibres as those having a 3:1 length-to-width can introduce identification ambiguities in some cases. In other cases, chemical or heat treatment of asbestos fibres may lead to problems in identification. "It is doubtful whether these definition problems will be solved quickly. More medical data are required to give guidance," Dr. Chatfield said.

He noted that the counting of fibres in a sample is subject to statistical fluctuations which affect the precision of the final result. In a sample of 100 to 200 particles, for example, a range in the measurement of about 30% on either side is avoidable. He also said that there is no international standard concerning the criteria for fibre counting, and opinions differ as to how to count arrangements of fibres (such as overlapping or split fibres). The common practice is to move away from complex clumps, but this usually results in a low count.

Finally, Dr. Chatfield discussed sampling techniques for building insulation material. He said it is important to obtain a sufficiently large number of samples since insulation is very inhomogeneous; that is, it is not evenly mixed. In fact, some samples may contain only mineral wool, a material commonly used in insulation. In 170 samples collected at an Ontario high school, 82 contained no asbestos at all and the remaining samples contained 1% to 40% asbestos. "By the collection of so many samples, it was possible to define regions of the building which required removal of insulation and other regions where no asbestos was present....On the basis of a few samples, it would have been possible to overlook the presence of asbestos in this building."

Dr. Chatfield noted that the mere presence of asbestos does not provide sufficient information to determine the amount of hazard; this depends also on the nature of the asbestos-containing material and how it has been used. Factors such as accessibility, presence in the air stream, condition of the material, and water damage are also important.

Dr. Chatfield said several techniques have been suggested for analyzing insulation; unfortunately, some inappropriate techniques have been used "with disastrous results." He noted, however, that the precision of the measurements is not the only important factor: it does little good to analyse a single sample to 1% accuracy, when the material may contain 40% asbestos in one location, 10% in the next,and 1% in the next. In such cases, more useful information might be obtained by analyzing a large number of samples with a simple and inexpensive technique than by analyzing a few samples with a highly-precise but more costly method.

He conluded by saying that many of the difficulties encountered in measurements in the last five years have been corrected, "but there are still quite a lot that have not been fixed yet."

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After Dr. Chatfield's formal presentation, there was an open question period.

Dr. Robert J. Uffen, professor of engineering and geology at Queen's University and a member of the Royal Commission on Asbestos, commented on the importance of technicians' expertise, especially with optical techniques, and asked what level of education technicians need and how long it takes to train them.

Dr. Chatfield said he has taught technologists to do the optical identification of insulation in about three months. The educational level is community college. He said the technician will still call him in for discussions on problem samples. However, this level of training is probably adequate to deal with 75% or 80% of the samples encountered. "We have easy samples and difficult ones."

Mr. Jack Bird is the National Representative at the Ontario Regional Office of the Canadian Union of Public Employees (CUPE). He asked whether it would be possible to establish a certification procedure for operators doing the testing. Dr. Chatfield said that certification does not exist at the moment, but it would be possible to start such a programme. He said there are only about four or five locations in Ontario where these sorts of measurements can be made. He said there are courses on electron microscopy, but they are mainly biological. "There's very little in the mineralogical field in this area."

Mr. Bird asked which is the common method used for air samples in public buildings: optical or electron. "It's been both," Dr. Chatfield responded. In cases where removal is underway, the optical methods give immediate and reasonably accurate results. In one Toronto building, the measurement was difficult and "we needed to go to electron microscopy. I think our opinion is that in a public building, optical microscopy is really not very good."

Finally, Mr. Bird inquired whether, in the case of operator error, there would be a tendency to over- or underestimate the particle count, and he asked how many air samples are needed for accurate results. Dr. Chatfield said that, with a well-trained operator, the results will encompass a range on either side of the correct value; however, an inexperienced operator may encounter

the problems outlined previously, which produce low results. As to the number of samples, Chatfield said it depends on where the samples are taken. More than one sample is obviously needed. "You get more accurate the more you take."

Mr. Bird asked: "But if you were checking a building for the presence of asbestos, how many samples would you recommend be taken in each location?" Dr. Chatfield said the question is difficult to answer. He said a half-dozen or ten samples might be taken. But electron microscopy rather than optical microscopy techniques would be used. In this case, they would be able to work to a detection level of .001 fibres per millilitre, which is below the Ministry of the Environment guideline of .04 fibres per millilitre.

Dr. Fred Hopton of the Ontario Research Foundation said that, in 1980, the ORF collected between 100 and 200 samples in many public buildings -- schools, hospitals, etc. -- with known asbestos-containing insulation in a wide range of conditions. He asked Dr. Chatfield to indicate the range of airborne concentrations of asbestos detected with electron microscopy. Dr. Chatfield responded that, in the case of fibres longer than 5 micrometers (on which Ontario's current guidelines-and its proposed regulations-are based), there was only one case where the number of fi-res exceeded the number found outside, and this was after an asbestos cleanup had been done. The general experience has been that the total fibres "are consistent with something we would find outside on the street. Air sampling has not generally given us very much of a result." The results have always been low, sometimes bordering on the lower sensitivity of the techniques used. "We usually find some fibres, but then you'll find some fibres wherever you take an air sample."

Mr. Eric Symes of CUPE, Local One, said that present measurement methods are not totally reliable. "The only real facts we have about asbestos are the many lives of workers that have been lost or critically impaired." He asked whether an effort should not be made to establish the lowest possible levels of exposure to workers while attempts are made to remove asbestos from the workplace. Dr. Chatfield said this was not within his area of competence, but "we do need to improve the methods of measurement."

Mr. Gyan Rajhans of the Occupational Health Laboratory at the Ontario Ministry of Labour asked whether cost comparisons had been made between polarized light (optical) microscopy and electron microscopy techniques for bulk sample analysis of asbestos. Dr. Chatfield said these comparisons had not been made because electron microscopy techniques are not really meant for bulk analysis. Mr. Rajhans asked what was the cheapest precise method for these analyses. Dr. Chatfield replied polarized light microscopy, which the ORF is doing at about $40 per sample. He added that the Foundation sometimes absorb the costs of using more sophisticated methods, which are infrequently used.

Dr. Chatfield said insulations are divided into a number of broad categories, and "You can pretty well characterize them on the basis of an optical examination." He said that if the polarized light technique were to be backed up with the X-ray technique, the cost probably would be more than doubled. He said the X-ray technique would probably cost $70 or $80 per sample; he added that this technique does not distinguish among the various varieties of amphibole asbestoses. "Discrimination between the amphiboles...is extremely difficult and can be erroneous....It can miss

material; so for routine screening, it's not a good thing...." In general, he said, "it's not really worthwhile going that route."

Dr. John L. Sullivan, Director of the Occupational Health and Safety Resource Centre at the University of Western Ontario, asked about the lack of correlation between phase contrast optical microscopy and electron microscopy. He said a great deal of money is being spent to tear out and/or replace asbestos,"and much of this is being done on false premises, I believe. In many cases, we've found on testing with optical microscopy that the counts are higher than [with] the electron microscope" -- much of this due, he suggested, to the presence of other fibres.

Dr. Sullivan said that most of the counts the Centre has obtained in public buildings have been very low; in cases where they have been high, this has been due to the other fibres. These sometimes cannot be differentiated by optical microscopy. Thus, he said, the Centre tries to dissuade people who want them to do counts using optical microscopy.

Dr. Sullivan also asked whether Dr. Chatfield's group had attempted to correlate the state of the asbestos with the number of fibres in the atmosphere. "We find that even when the asbestos is in rather poor shape, the fibre counts in the atmosphere are very low." He said he did not believe this was due to the period of time they happened to take the measurements; the counts would have been low at any time. Yet some medical experts have "more or less thrown doubt on the practice of taking air counts at all."

Dr. Chatfield commented that, with respect to air sampling in buildings, problems with airborne fibres are most likely to occur with the amosite compositions, loosely-held materials that become airborne readily. The chrysotile asbestos tends to be rigidly attached to heavier material and it doesn't appear to get airborne readily.

He said his group also tries to dissuade the taking of optical fibre counts inside buildings "because we don't know how to interpret them. Indeed, I don't think we really know how to interpret the electron microscope count, apart from saying: This is as low as we can go and we're going to find one or two background fibres, which we will also find outside the front door." He said Dr. Sullivan may be correct in saying that, in some cases, removal may have been done when it was not really necessary.

Mr. G. Peter Robson of Dupont Canada Ltd. commented on monitoring for airborne fibres in industrial environments and tearing out old insulation, He said Dupont has found that insulation installed many years ago may contain at least two different types of asbestos. When airborne sampling is done, there is the problem of determining how much of each type is present. In addition, there is the fact that exposure limits are coming into effect that specify different values for the different asbestos forms. He asked Dr. Chatfield to describe the techniques and costs involved in distinguishing between two types of asbestos occurring simultaneously in an airborne sample.

Dr. Chatfield said that when you know what the material is, many identification problems don't apply. It is one situation to identify long, thin fibres from unknown sources in the general environment; it is another situation when you have a known insulation containing two asbestos types and you want to find out what's in the air sample. He suggested a technique using the scanning electron microscope,which is the quickest, but said it would be somewhat more expensive than using an optical microscope. This would provide a means of discriminating between different asbestos types.

Mr. Robson asked about changes in some of the varieties of asbestos caused by exposure to heat. He said that in some industrial sites, insulation has been in contact with hot metal for many years. Dr. Chatfield said chrysotile will gradually degrade under these circumstances and samples of this material might produce a zero result. Amosite will not do this.

Mr. Charles Mallory of STOP (Acid Rain) asked about the difficulties of measuring asbestos in water samples, and about the reliability of such samples compared to air samples.

Dr. Chatfield said the basic difference between air and water samples relates to the size of fibres likely to be encountered and the form they are in. Fibres in air samples are highly aggregated in random directions, the result being rather like "a bird's nest floating around." This creates counting problems. "With water this is not so common and the fibres are very much smaller." There have, however, been problems in handling the water samples and getting reliable results. The ORF has reviewed the reliability of various methods, and Dr. Chatfield said there was good statistical agreement between the ORF's Canadian drinking water survey and results obtained by U.S. Government labs. "I think the methods now are much better than they were five years ago, but air measurements are probably still somewhat less reliable than water because there's not that much work been done on the air sample as yet."

Dr. Dave Verma of McMaster University's Occupational Health Program asked about asbestos contamination in nickel-containing areas. As an example, Chatfield said the drinking water in Thompson, Manitoba has chrysotile from local rock.

Dr. Verma asked about a low-temperature procedure to eliminate problems with non-asbestos fibres in a sample. Dr. Chatfield noted that the objective is to make a lot of measurements cheaply. The use of additional techniques increases the amount of effort and, if optical measurements provide adequate answers, there is not much point in going to added techniques.

Dr. Verma commented that an infrared technique has given good results in identifying and quantifying fibres. Dr. Chatfield said a number of labs are using infrared techniques.

Ms. Wendy King, who is with the Canadian Centre for Occupational Health and Safety (but who stated she was asking her questions on a personal basis), asked about the testing of a building in Hamilton and the effect of human

activities. She asked (a) if care is taken to ensure that normal human activity is going on during testing; (b) if human activities interfere with the accuracy of the tests and (c) if testing in a gymnasium was done while students were playing basketball.

Dr. Chatfield said that human activity increases the airborne concentration of fibres, due to re-suspension. There are three situations: material falling from the ceiling, resting on the ground; and being disturbed and re-suspended by humans. "Our practice is to try to do the sampling during normal activity within the building." However, problems may occur because the pumps are noisy and this can disturb occupants, who sometimes resist this method of testing. He said human activities probably do not interfere with the measurements. "You're trying to get a sample which is representative of the air in the building as people are living in it."

Ms. King asked whether it wouldn't be impossible for a group of teenagers playing basketball not to interfere with the machines. "Practically impossible, I would think," Dr. Chatfield responded. "So the testing that was done in that room where there was no human activity wouldn't necessarily be representative of what people were breathing," Ms. King asked. Dr. Chatfield said that the readings would be lower than they would be if people were moving around.

C. The Health Effects of Asbestos and other Hazardous Substances: Assessing the Quality of Scientific Evidence.

C.1 Address by Dr. David L. Sackett, M.D., M.Sc.Epid., Professor of Epidemiology and Biostatistics, Professor Medicine, McMaster University on "A Review of Diagnostic Tests for Causation"

Commissioner Dr. J. Fraser Mustard chaired the meeting's second agenda item on the health effects of asbestos and other hazardous substances and assessing the quality of scientific evidence. He first introduced Dr. David Sackett, who is an internist, clinical epidemiologist, and currently professor of clinical epidemiology and biostatistics and of medicine at McMaster University in Hamilton, Ontario.

Dr. Sackett's interests include the design, execution and interpretation of randomized clinical trials, the assessment of the diagnostic process, and the study of the clinical course and prognosis of human disease.

The title of Dr. Sackett's address to the Commission's public meeting was "A Review of Diagnostic Tests for Causation." That paper appears as Appendix B to these Proceedings.

Dr. Sackett's talk focused on the problem of establishing cause and effect links between asbestos and disease, and on the possibility of developing diagnostic tests to determine causation.

He pointed out that claims of causation abound in medical journals and the media -- claims, for example, about the links between cigarette smoking and occupational lung canacer; between malfunctioning brown fat and obesity; between colour-blindness and driving accidents; between snow shovelling and heart attacks, and so forth. "The diagnostic test of

causation is an issue which faces not only medical scientists and environmentalists, but faces each one of us every day as we read newpapers and as we listen to other media as they present the latest scare about what we breathe, eat, sleep or smoke..."Dr. Sackett said. During the past two decades, diagnostic tests have been developed to help not only specialists but also the public sort out claims for causation and separate cases of true causation from those that merely represent association.

Dr. Sackett discussed several diagnostic tests:

(a) True experiments in humans: studies in which humans are randomly allocated (by a system analagous to tossing a coin) to receive or not to receive exposure to the agent believed to be the cause of a certain effect. These people are then followed up to see if the effect occurs. This provides the "best evidence we will ever have about causation, "Dr. Sackett said.

Such "randomized trials" have become the standard approach in determining the benefits of therapy and account for "major therapeutic advances in the Western world in the last 20 years concerning the treatment of major killing disorders such as coronary heart disease, hypertension (high blood pressure), a host of infectious disorders,and other chronic diseases."

However, the technique -- and the random allocation of exposure -- is clearly questionable when the effect of a potentially harmful agent is at issue. In cases where there is concern that exposure will be dangerous, judgements must be made on the basis of other types of evidence. In these cases, great emphasis is placed on the second diagnostic test, the strength of the association between purported cause and effect.

(b) Strength of association: how much greater is the risk of developing the outcome with, as opposed to without, exposure to the possible causal factor? Attempts to measure strengths involve two differenct sorts of research strategies -- the "cohort" study and the "case-control" study.

The cohort approach involves identifying two groups of people, one which is exposed to the causal factor (for example, in their work) and one which is not exposed, and following them over time to see if the effect occurs. The higher the strength of association, the more likely is causation. Dr. Sackett said this is a powerful technique but it has limitations -- particularly when the effects being looked for are rare and/or late in their occurrence.

For this reason, the second, or "case-study," approach is often used. Here again, two groups are chosen: one in which the effect of interest has already occurred and another in which it has not. They are then investigated backward in time to determine their prior exposure to the possible causal factor.

Of the two methods, the cohort study is less susceptible to biases in sampling, measurement, analysis, and interpretation and "therefore provides much sounder evidence of causality." But if the effect of interest takes many years to occur, and if it occurs only rarely, this technique could be a very expensive and time-consuming technique and "we may want to make a practical decision long before its results would be known." Case control studies, on the other hand, are quicker and less expensive "because nature and causation has already forced the events to occur and one simply tracks them backwards." Thus, this method is well-suited to studying rare and/or long-term effects. However, many biases can occur that lead to incorrect conclusions about causality; worse, in many circumstances, it may not even be recognized that the biases have occurred. Unfortunately, many of these biases tend to increase spuriously the measure of risk.

Dr. Sackett then illustrated how the cohort method works by referring to a study on the link between smoking and lung cancer. A smoking and a non-smoking group of 100,000 people each were followed forward in time; in a typical year, 110 of the smokers but only 10 of the non-smokers developed lung cancer. Thus, smokers were about 11 times more likely to develop lung cancer than the non-smokers.

In his prepared text, Dr. Sackett referred to another technique: the "case-series" method, in which it is simply reported that a certain percentage of people who demonstrate the effect being studied had been exposed to the possible causal factor. No comparison group is studied. Dr. Sackett said this method is weaker than the others and is best used as a spur to using more powerful techniques.

In his paper, Dr. Sackett concluded that the randomized trial "is the strongest and usually can be trusted.... A cohort analytical study, although weaker...is always preferred over a case-control study and can sometimes be trusted....The case-control study is a weak design and has often led to erroneous conclusions....However, for some extremely rare disorders...we may have only case-control studies to go by and may be forced, however reluctantly, to trust them. Finally, it is not possible to tell whether any given case series can, all by itself, be trusted on an issue of...causation. Thus, if other, stronger evidence is available, such case-series should be passed over." (See Appendix B, p. 4.)

(c) Consistency: Do different investigators working in different locations using different research strategies come up with consistently similar conclusions? Using the smoking example again, Dr. Sackett pointed out that more than 30 case-control studies and nine cohort studies have produced much the same results.

(d) Temporality: assurance that exposure occurred before the occurrence of the effect being studied. Usually, this is reasonably straightforward but sometimes it is not. (One disputed example is the relationship of mental illness to living in the core of large cities.)

(e) Dose-response relationship: a powerful means of attempting to demonstrate causation. The issue here is whether higher and/or more prolonged exposures to the purported causal factor leads to earlier, or more frequent, or more severe outcomes. Dr. Sackett said this is perhaps the most convincing evidence for the link between smoking and lung cancer. For example, even those who smoke 10 cigarettes or less a day are five times as likely to get lung cancer as non-smokers. Moreover, the rate of risk rises: those who smoke 20 cigarettes or more a day have a 20:1 risk and the two-pack-a-day smoker has a risk of 36:1. Alternatively, risks decline among smokers who give up the habit. Thus, risksare clearly related to the dose of smoking.

Dr. Sackett elaborated on the dose-response issue, responding to the following questions: Does the presence of a dose-response relationship prove causation and, on the other hand, does its absence disprove causation? Do thresholds exist below which variations in the dose make no difference? Are there "safe" thresholds? (Dr. Sackett discussed these issues in general terms; the following speaker, Dr. David Muir, related them specifically to asbestos.)

Dr. Sackett said that the absence of a dose-response relationship "certainly would support a non-guilty verdict." But several circumstances, often related to insufficient quality of data, can obscure a dose-response relationship and make it appear not to be present, when in fact causation does exist. For example, if the measures of exposure or dose are not precise, this would lead to misclassification. "Individuals who are exposed to high doses would appear to be exposed to low doses; individuals exposed at low dose would be misclassified as having received high doses....So the quality of evidence is a key issue."

Other factors can obscure the true dose-response relationship. Four examples follow:

(i) It is possible that only a minority of the people exposed to the causal factor were really susceptible "and these individuals might be swamped by a majority of non-susceptible individuals."

(ii) If too narrow a range of doses or exposures is examined, important aspects of the dose-response relationship may not be measured.

(iii) There might be multiple sources of exposure agent which may not all be measured when determining dose.

(iv) The dose-response relationship can examine only a single cause, but many human illnesses have multiple causes.

As to whether the existence of a dose-response relationship proves causation, Dr. Sackett said it certainly supports a causal conclusion, but does not alone provide proof.

He warned also that an apparent dose-response relationship may in reality be a "marker" relationship; that is, the occurrence of an effect correlates with exposure or dose, but is not caused by it. An obvious example: the dose-response relationship between the use of matches and lung cancer risk.

"Yet, of course, it is not the matches themselves that cause the lung cancers -- it is the cigarettes that are lighted by those matches. So...one needs to be concerned about whether one is simply measuring a marker rather than a true causal agent."

He said that statistical methods can be used to deal with these "confounders" of the dose-response relationship.

Dr. Sackett next turned to the issue of thresholds below which variations in dose make no difference; or, in other words, whether "safe exposures" exist. Here again, the quality of data often causes limitations. If the response to a dose is rare and/or delayed in time, a threshold may appear to be present simply because the study has not gone on long enough.

Dr. Sackett also noted that there is a major problem in interpreting the graphs showing dose versus response, and he outlined some of these difficulties. The graphs typically show increasing response along the horizontal axis, with zero-dose and zero-response meeting at the left-hand corner.

Often there is no actual response data for the low-dose, low-response corner of the graph, and Dr. Sackett warned of the danger of simply extrapolating or "drawing a line" through the data back towards the low-dose area. "We simply do not know what might occur, in term of dose versus response, below any set of actual observations and extrapolation back towards zero is fraught with all kinds of uncertainty."

Dr. Sackett said that if data exist for the low-dose, low-response corner of the graph, it may be possible to establish whether a threshold exists. (For example, if a response line reaches zero before the dose reaches zero, there is a threshold below which changes in dose make no difference. Alternatively, if a response line hits zero-dose before or simultaneously with reaching zero itself, there is no threshold, and any increase in dose will cause an increase in response.) However, Dr. Sackett warned that careful attention must be paid to the quality of data, particularly in the low-dose areas, in making these judgements.

Dr. Sackett then briefly outlined the remaining diagnostic tests:

(f) Biologic sense: using existing knowledge about the responses of tissues, cells, organs and organisms and whether or not the effects observed are consistent with the understanding of human biology.

(g) Epidemiological sense: the distribution in time and space of exposure and outcome; for example, correlating sales of cigarettes in each of the states in the U.S. and the occurrence of lung cancer in those states. The correlation (.81) is one of the highest ever observed, Dr. Sackett said.

(h) Specificity: Is this a single-cause, single-effect circumstance? If so, "we're in relatively good shape, both in terms of sorting out causality and in terms of preventive measures." As an example, he cited the study of 100,000 smokers and 100,000 non-smokers. He said that more than 90% of the lung cancers can be explained on the basis of cigarette smoking "so that this cancer would virtually disappear if cigarette smoking stopped."

(i) Analogy: Is there a similarity to a previously-observed causal relationship? The case of cigarettes and lung cancer related to earlier work on other inhalation disorders, for example that chimney sweepers had high rates of skin cancer on areas of their body where soot collected.

In closing, Dr. Sackett discussed two issues that arise in clinical medical situations in which doctors must decide whether to use certain therapies or diagnostic procedures, or recommend lifestyle changes to patients. The certainty of causation is only one issue; the other has to do with the consequences of alternative courses of action: in other words, weighing risks and benefits, once it has been established that causation is likely.

For example, a few years ago, it was suggested that a drug commonly used to treat hypertension (high blood pressure) might be linked with breast cancer. The proof of causation was not great, but there were alternatives, so use of the drug was discontinued. On the other hand, it appears that oral contraceptives can cause various heart and circulatory problems, but the consequences of alternative courses of action are such that the approach has been to discontinue use primarily among patients who are high risks.

C.2 Address by Dr. David Muir, Director, Occupational Health Program, McMaster University on "The Health Hazards of Asbestos: Assessing the Quality of Scientific Evidence

Commissioner Dr. J. Fraser Mustard next introduced Dr. David Muir who is the Director of the Occupational Health Program at McMaster University, Hamilton, Ontario. Prior to this appointment, Dr. Muir served as the Director of the Institute of Occupational Medicine in Edinburgh, Scotland. Dr. Muir is a respiratory specialist and consultant. His main research interest is the behaviour of sub-micron aerosol depositions in the lung.

The text of Dr. Muir's speech to the Royal Commission on Asbestos public meeting appears as Appendix C to these Proceedings.

Dr. Muir started his talk by stating that there is no doubt of the relationship between the duration and intensity of exposure to asbestos and the health risk. "The evidence for this is overwhelming and is virtually self-evident." Thus, unnecessary exposure should be avoided and "good housekeeping" practices designed to decrease exposure both in the workplace and in the general environment are desirable.

He said noted airborne asbestos concentrations in public areas are much lower than those occurring in asbestos manufacturing industries and expressed doubts about the practical value of airborne monitoring, since measurements of airborne asbestos are extremely difficult to take and interpret.

"I do not think it is possible to show in rigid mathematical terms that inspection of the workplace or inspection of the public place is less helpful than taking airborne fibre concentrations. The difficulties with the airborne measurements are that they are technically very difficult and there are only very few centres that can be trusted to do it accurately.... Since .... inspection of a building, taking account of the type of material and its accessibility...can be shown to be so overwhelmingly more useful from the practical point of view, that is really why I'm on record as saying the balance has to be in favour of visual inspection rather than the monitoring."

He said, however, that monitoring is useful and, as a research field, "needs looking at."

Dr. Muir then pointed out some difficulties facing those required to frame policy. A major one is the fact that disease now attributed to asbestos -- particularly mesothelioma -- had its origins in exposure to asbestos many years before. There is little evidence concerning environmental conditions two to four decades ago and, while there is little doubt dust levels were higher, it is questionable whether the estimates made are adequate for current mathematical modelling studies.

A second major problem is to separate the risks associated with different types of fibres. If it is accepted that there are specific effects due to different fibre types, then it becomes important to identify the types of asbestos used in the past in individual companies. This can be surprisingly difficult to do.

Dr. Muir said there appears to be strong evidence that health risks depend on specific industrial situations; for example, the size distribution of fibres in an air sample varies according to the industrial process. The practical and economic importance to Canada is considerable. "The apparent lack of health effects claimed for chrysotile workers in Quebec at relatively high levels of exposure is remarkable. We need to know why there was such a large contrast in their mortality experience compared to insulation workers."

Asbestos-related diseases generally appear not only years after first exposure, but many years after exposure has ceased. Thus it is necessary to study the health experience of workers in relation to exposure levels and the time since exposure first occurred. Dr. Muir said some of the asbestos literature does not follow this concept, adding that the Commission could do a valuable service by analyzing the acceptability of currently available information of this kind.

He said also that the reliability of early estimates of exposure should be examined. Measurements were often only approximate and, when they did not exist, long-time employees were asked what it was like in "the old days." Thus, there is great variation in the estimates of previous exposures. Some people argue that previous dust levels were very high and that current levels are small by comparison and therefore relatively harmless. Others argue that previous levels were not that high and that current levels are unacceptable.

Mindful of these limitations, Dr. Muir then proceeded to give conditional answers to the questions posed by the Commission regarding dose-response curves and thresholds for asbestos exposure.

He concluded that the dose-response data for lung cancer indicate that "reducing dust levels is likely to have health benefits and that low-level exposure is likely to result in low-level mortality."

He noted the rather "remarkable" evidence that there does not appear to be an increase in deaths among workers in chrysotile mines exposed to high fibre content, while measureable health effects have occurred among workers in fabricating and insulation industries, who are exposed to much lower levels of chrysotile. (He said, however, that in the mining industry, "regretfully, the method of measurement was not one which enables us to transfer easily or with any confidence to the...method used outside the mining industry.")

He said it would be unwise to assume that, since these moderate chrysotile levels do not cause trouble in the mining industry, they do not cause trouble in the fabrication industry.

As for mesothelioma, there is little evidence to determine the dose-response relationship, but it appears certain that heavy exposure is more likely to cause the disease and that this effect relates to the type of fibres involved. No zero effect has been identified, but this does not mean there is no lower threshold.

"The real difficulty...is lack of reliable data concerning the health effects of low level exposure....Firm predictions are unlikely to be well-founded."

The uncertainties make it difficult to estimate how much health improvement might result from establishing policies to reduce asbestos exposure, Dr. Muir said. However, he said it is relatively easy to control gross airborne contamination in industry and visual inspection of, and rapid attention to, the more obvious sources of contamination in public areas are likely to be beneficial. He said airborne sampling results are less likely to provide a sound basis for action. He concluded that long-term observations are needed to measure the levels and types of airborne fibres.

Dr. Muir commented that, in establishing regulations or controls, it is important to remember that any actual health improvements will be related to the extent to which the regulations are complied with on a day-to-day basis. A lower standard of control may provide greater health benefits than a more rigid one, if the former achieves 95% compliance, and the latter only 50%, for example. "Defining very rigid standards which are ignored appears to be an exercise in futility for all concerned."

Finally, Dr. Muir said it is very important to do additional research to determine levels of exposure and, especially, to provide detailed descriptions of the types of fibres in the air, since it may be that different types cause different health effects.

He concluded that any health benefit to be derived from regulations will depend on day-to-day compliance with the regulations in the workplace. "If I were in the asbestos industry, I would rather have 95% confidence that no worker is going to be exposed to greater than a certain fibre level, rather than 50% confidence in relation to some more rigid level. I do not think we can avoid this decision that, in getting standards, one has to take account of how readily and how easily they can be measured, and what is the likelihood that they will in fact be observed...It seems to me that defining standards which are subsequently ignored is likely to be an exercise in futility for everybody."

In response to the question: "is there a safe level?," he said the "evidence does not allow us to define that. Equally, I doubt if it allows us to say that such a level could not exist." However, he said, certain decisions are possible. If asked whether the evidence is sufficiently overwhelming to close the asbestos mines next week, the answer would be that this decision would not be supported by the evidence at the moment. However, it would be possible to decide whether the use of asbestos in particular situations would be justifiable; whether there are reasonable alternatives; and whether the counting techniques used to protect people were really adequate.

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A question period followed the addresses by Drs. Sackett and Muir.

Mr. Eddy Cauchi, a former asbestos worker, said he did not doubt that the equipment used for air monitoring is the best available. But, in reference to the discussion of costs, he said that cost has nothing to do "with human lives and the public's health. So if we're going to look at what it's going to cost us, or the company, or the government, I think that's a backward step. The cost is the health."

He asked Dr. Muir whether it's true that "workers who have asbestosis have nothing to do with smoking."

Dr. Muir said it's generally believed that asbestosis -- the fibrosis of the lung -- is not related to cigarette smoking. But even this statement is not as easy as it sounds; there is some evidence that cigarette smokers do have an increased tendency to develop fibrotic changes. "The evidence is disputed. The assumption at the moment is that these two conditions are not related." He said this goes for mesothelioma as well.

Mr. Cauchi said that 61% of the miners in Quebec tested in 1978 were found to have abnormal lungs from asbestos dust. He asked how many workers' deaths due to asbestos have been blamed on other diseases, because of a lack of awareness about asbestosis in Ontario. "Am I correct to assume the mortality rate is misleading?"

Dr. Muir said he was familiar with the Quebec data, but he didn't know the total mortality from asbestos in Ontario. "I'm not sure any figures have been published." He said there are data concerning mesothelioma, asbestosis and compensation for lung cancer.

Mr. Cauchi said he disagreed with Dr. Muir's comments about dose levels. He said that only a "slight dose" caused the death of a school board employee in Scarborough. "So the dose actually has nothing to do with it and neither does the length of time the worker works in the asbestos industry. This was proved."

Dr. Muir disagreed. He said he was familiar with the Scarborough case and similar ones, but "overwhelmingly, the majority of cases have come from heavy exposure with long exposure. The evidence for that is sufficiently dominant...."

Mr. Cauchi asked whether, in Ontario, there are only guidelines, but no laws or standards, governing airborne concentrations.

Dr. Muir said he believed the law is to be implemented shortly, but at present there is a guideline.

"There are no court cases or action the government could take if the industry exceeded the level in the air?" Mr. Cauchi asked.

"That is my interpretation at the moment," Dr. Muir responded, although he expressed reluctance to speak for the government.

Mr. Cauchi asked Dr. Sackett where he got the data for the study on cigerette smoking and lung cancer. Dr. Sackett said the data came from about 150 studies done all over the world. Mr. Cauchi asked if McMaster University had done any studies in Ontario on lung cancer and smoking, and Dr. Sackett said yes.

Mr. Cauchi then said there were three widows in the audience and "their husbands never had a smoke in their lives, and they were never questioned on this study you're talking about."

Dr. Sackett said this illustrates the point made earlier about disorders like lung cancer that have multiple causes: it is to be anticipated that individuals never exposed to one cause, but heavily exposed to one of the others, would develop the disorder.

Mr. Cauchi then cited a 1977 report from New York that there is currently no way to determine a safe level of asbestos. He went on to quote a statement made in 1980 by an official of the Occupational Health Branch of the Ontario Ministry of Labour, who said that .1 asbestos fibres per cubic centimetre of air should be the standard. He then quoted a Dr. Parkinson, saying that there was a chance the standard wouldn't go through because large corporations that mine or utilize asbestos have complained that they would be forced out of business. He asked Dr. Muir if it is possible to achieve the level of the proposed standard, or zero level.

Dr. Muir said .1 fibres per cubic centimetre is usually taken as the lower limit of practicable detection for optical microscopy; conceivably, it could go below this if electron microscopy were used.

Mr. Cauchi then turned to the issue of the different types of asbestos. He referred to a 1975 article in the New Scientist magazine suggesting that fibres of less than 5 microns in diameter may produce tumors, regardless of the type of fibre.

Dr. Muir said that animal research by the U.S. National Cancer Institute demonstrated that it is possible to produce mesothelioma-type tumors with "fibres with a broad range of chemistry, including materials outside the asbestos range." He said there are asbestoform minerals other than the ones he mentioned in his talk.

Mr. Cauchi asked Dr. Muir whether he was aware of a report in the Harvard Medical School Health Letter saying that every possible way should be found to remove asbestos from the human environment. Dr. Muir said he was.

"So, therefore, you have to agree with me that exposure has something to do with it -- that the safe level is nil; there is no safe level of asbestos fibres in the air."

Dr. Muir said he had already made it clear that it can neither be proved that there is or is not such a safe level.

Mr. Mallory from STOP (Acid Rain) was the next questioner. He asked Dr. Muir if he was satisfied that an adequate amount of research is currently being done into the relationship between very low levels of exposure in the general environment and incidence of disease, so that we may resolve the problem caused by the current lack of evidence. "If not, since this area's been identified as a problem, isn't this a priority?"

Dr. Muir said that anyone in the academic research field is likely to say that funds are not adequate. "There quite clearly are research questions that need answering." One of the major problems, he said, is that asbestos is "a very complicated substance: and very few people who haven't worked with it realize just how complex it is -- including university researchers. He said one of the problems is to create an independent group of people to collect the needed information, who have the energy, time, and initiative to do the job and are free of pressure from government, unions, and industry. "You need a bunch of people; you don't need one odd individual hither and thither."

He said decisions about the use of asbestos will have enormous economic implications for Canada and, if we are going to continue using this material, "there's no doubt at all that a very considerable effort has got to be put answering some of these research questions. At the moment, I don't estimate that effort is being put in."

Mr. Lionel Ferguson of the Toronto Board of Education asked Dr. Muir whether schools should be considered as a "unique situation" and whether the Commission "should address itself in particular terms to schools and young people."

Dr. Muir said this reinforces his view that each environment should be considered separately, and "that what may be satisfactory in one situation may not necessarily be alright in another." He said that fibres inhaled by school children "will be there for life" -- certainly in the case of the amphiboles, although probably less so in the case of chrysotile. "But I think that the community would be on solid ground to make a decision that whatever is good enough in a building such as this, should be that much more rigidly controlled where children are concerned. It's difficult to be very scientific, but it seems to me a reasonable decision that schools should be handled more carefully than other buildings. I'm not trying to say that other buildings should be less carefully handled, but one's got to start somewhere." He said that one would be on "unassailable grounds" to say that if children are present and are using certain areas for prolonged periods of time, "then they merit more attention than perhaps the rest of us. I'd certainly want my own children to be treated in that way, and I think this is a not unreasonable approach."

Mr. Gyan Rajhans of the Ontario Ministry of Labour's Occupational Health Laboratory made a few comments on standards and guidelines. He said that while it is true that no standards have yet been promulgated, the Ministry has worked with guidelines for many years, and it has the jurisdiction and authority to enforce them although they "still are construed as guidelines." He said they have been enforced in the mining and construction industries. "We have had some very good results, and we have published papers based on what results we obtained by enforcing the guidelines."

He asked if it is difficult, in epidemiological studies, to find a cohort group of people for asbestos -- in other words, a population reasonably assured to have had no asbestos exposure at all -- since asbestos is ubiquitous and anyone can be exposed at some point in his life.

Dr. Sackett suggested that it might be possible to find "geographic isolates": individuals "who would be living in such different environments from our own, that not only would they not have been exposed to asbestos, but they would not have been exposed to all sorts of other agents and experiences." However, this might produce "fairly formidable" problems of analysis.

Dr. Muir added that asbestos in the normal environment comes not only from industrial processes, but, in many regions of the world, from natural sources. He said there are large rock outcrops in the United States and Europe, and all populations there are exposed to some extent. "So it's not quite so easy to find a completely non-exposed population as you might think."

Mr. Ray Gibson of Ontario Hydro said he works with employees who are potentially exposed to asbestos and who have routine X-rays. He asked whether there are any cohort studies relating the condition known as plural plaque (defined by Dr. Muir as a visible abnormality that can be seen on the X-ray film) to mesothelioma. "In time, is there any tie-in between the two?

Dr. Muir said there's no evidence at the moment that plural plaque is a pre-malignant condition. The problem is that plural plaques are, by definition, caused by asbestos exposure; therefore, those who develop the condition are more likely also to develop mesothelioma. "If you took all the people with plural plaques, you'd find that there was a greater incidence of mesothelioma than in the normal population. But if you standardized for dose, there is no evidence at the moment that the plural plaque is a pre-malignant condition," Dr. Muir said.

Mr. Edward Lescisin, a student of environmental studies at the University of Toronto, asked Dr. Muir a question relating to dose-response curves. "You mentioned that often the graph of the relationship is extrapolated back through the origin as a matter of convenience, even though the response has not been determined at these unmeasured low dose levels. This implies a zero threshold relationship. Have you eliminated the possibility that certain members of the population are susceptible to asbestos-related diseases that would be associated with a break-point relationship?"

Dr. Muir said he'd tried to avoid the question of the susceptible individual because "I think this is a red herring. I don't think this is a useful arguement." With regard to drawing a straight line back through zero on the dose-response graphs, he said this line "cannot be shown to be any worse than any other line," but said scientists are really "not entitled to do that." They should draw the line to correlate the points higher up on the graph that indicate the range of exposures actually measured and "then quietly rub [it] out further down...The evidence doesn't support the suggestion that you should take much notice of where it is lower down."

Mr. Eddy Cauchi said the difficulty workers have with guidelines is that the government can't take legal action when the guidelines are broken. "For 25 years that I worked at the Johns-Manville corporation, there were guidelines....But during the 25 years -- and I have reports of government testing -- there were many times that guidelines were broken and I don't ever recall that the company was ever charged." In reference to Mr. Rajhan's comments, Mr. Cauchi said he had a 1972 report "when he left three orders... he told the company to clean up their act in certain places and then when he came back these weren't clean -- in fact, he reported to the Minister. There is no sense leaving any orders because they don't listen to you anyway."

Dr. John L. Sullivan of the University of Western Ontario asked Dr. Muir if there were any statistics available indicating the total number of people in the population who have asbestos bodies (described by Dr. Muir as a "long fibrous thing covered with a protein." He said not all such bodies are contained on an asbestos fibre.)

Dr. Muir said there have been many surveys on this. The figures vary depending on the type of environment; in large urban environments, it is expected that 50% to 60% of the normal population have asbestos bodies. He said it appears that the majority of asbestos bodies in the lungs of people in the general population are based on amphibole fibres "and most of us, we've all got some in our lungs." Although the chrysotile fibres are more present in the atmosphere, most of them seem to disappear.

Dr. E.K. Fitzgerald, Medical Officer of Health for Scarborough, pointed out that for various causal factors some calculations suggested that it may take hundreds of years for a disease to occur. He asked Dr. Sackett: "Does that constitute a threshold, when the length of time is two or three times a normal life?"

Dr. Sackett answered by reference to the cigarette/lung cancer example. Exposure is sometimes given in terms of pack-years -- equivalent to smoking a pack of cigarettes a day for a year. He said this is simply a way of putting together two different factors: intensity and duration of exposure. If someone smoked only a little, "one might have to go to pack-years of exposure much greater than the normal human lifespan....You may wind up with durations of exposure that biologically don't make a whole lot of sense."

Mr. Jack Bird of the Canadian Union of Public Employees asked Dr. Muir if it was safe to say that there is no way of determining acceptable levels of exposure; that such levels are determined through trial and error, and whatever levels are established may not be proved correct or otherwise for a decade or two.

Dr. Muir said he had been meticulous in avoiding the phrase "acceptable levels." He said it's correct to say there is no method of predicting the long-term effects of any given level, other than by trial and error. He said he suspected this is true not only for the very low levels found in the general environment, but also in the workplace. "Whatever level is to be used in Ontario, I suspect it will be based on the best information available at the time. I don't think one would be surprised if at some later date, there were cogent reasons for changing it. I think that this approach is good with all so-called levels -- if they are regarded as sliding data to be used and modified without hesitation when new information comes along. Perhaps if one could only persuade government departments to admit that yesterday's conclusions are superceded by today's conclusions; I see no harm in admitting that at all....Whatever level for public buildings is now used, I wouldn't hestitate to change my mind next week if new information comes along."

Mr. Bird then questioned the Commission: we've heard about the economic difficulties of reducing the levels to what we would consider acceptable. Has there been any consideration given to determining the medical cost to the victims of asbestosis and mesothelioma?

Dr. J. Stefan Dupré, Chairman of the Commission, said the Commission will be looking closely not only at the question of direct medical costs of illness related to asbestos, but at indirect costs: those social and other costs borne by individuals and their families as the burden of ill health created by environmental factors. "That will be one of our toughest tests as a Commission."

D. Frameworks for the Regulation of Hazardous Substances

D.1 Address by Dr. Steven Kelman, Professor, Kennedy School of Government, Harvard University, on "Frameworks for the Regulation of Hazardous Substances in Jurisdictions Outside Canada"

The Commission's Director of Research, Dr. Donald N. Dewees, chaired the meeting's third and final agenda item on frameworks for the regulation of hazardous substances. He introduced Dr. Steven Kelman, who is an Assistant Professor of Public Policy at the Kennedy School of Government, Harvard University, Cambridge, Massachussetts. He is currently on leave from the School and is working at the Federal Trade Commission's Bureau of Consumer Protection in Washington, D.C. Dr. Kelman is the author of the forthcoming book entitled, Regulating America, Regulating Sweden: A Comparative Study of Occupational Safety and Health Policies. This book is being published by MIT Press in February, 1981.

Dr. Kelman's address to the second public meeting of the Royal Commission on Asbestos focused on "Frameworks for the Regulation of Hazardous Substances in Jurisdictions Outside Canada." He talked about the United States, Sweden, and the United Kingdom.

The text of Dr. Kelman's speech appears as Appendix D to these Proceedings.

Dr. Kelman prefaced his remarks by noting that the newly-elected U.S. President Ronald Reagan has not been a strong supporter of federal environmental, health and safety regulatory agencies. "This is an anxious time to be a regulator," he said, adding that one of the themes he planned to develop related to the growing political controversy throughout the Western world on the regulation of hazardous substances.

Dr. Kelman began by comparing the framework for occupational health regulation in Sweden and the United States. There was a large upswing in government attention to occupational health and safety at the beginning of the 1970's throughout the advanced industrial Western countries. The U.S. Occupational Safety and Health Act was passed, which set up the Occupational Health and Safety Administration (OSHA). In Sweden, where there already existed a national government presence prior to the 1970's, there was an increase in attention and resources devoted to occupational safety and health. The Workers Protection Law of 1949 was strengthened and then replaced by a new Work Environment Law in 1976. In England, where there had also been previous efforts, the Health and Safety At Work Act was passed in 1974.

The increased attention to this field was "probably most dramatic in the United States" in that previously, all efforts had occurred almost exclusively at the state level. So OSHA was "part of a very dramatic expansion" in the federal role in American life, particularly in the area of health, safety and environment.

Dr. Kelman said it was interesting how quickly this happened. For example, the Clean Air Act amendments of 1970, which dramatically expanded the federal role in environmental matters, were passed "very quickly and almost without opposition in Congress."

The overall political environment in Sweden is very different from that of the United States; Sweden has a stronger labour movement and the politics are further to the left. This provides an interesting backdrop for comparing health and safety legislation in the two countries. Dr. Kelman said the statutes themselves are more similar than different, but there are some relatively important differences. However, the similarities in the implementing regulations and agencies are quite striking. (However, he said asbestos is something of an exception.)

Dr. Kelman then compared Swedish and U.S. statutes. Both establish "a rather vague but rather strict standard of protection" for working people that the regulations are supposed to attain. For example, the U.S. Act sets a goal that, to the extent feasible, no worker should suffer any ill effects from exposure to chemicals even if he works with the chemical for an entire lifetime. The Swedish Act says that the work environment should be "acceptable, considering the nature of the work involved and the social and technological progress in society at large."

Since this language is relatively vague, the agency implementing the law has a great deal of discretion in the level of protection that workplace health regulations embody. "It's not all laid out in the statute." The regulatory agencies in both countries are allowed to establish threshold limit values for exposure to chemicals having the effect of law. They are also allowed to require monitoring of hazardous substances in the workplace, medical exams for exposed workers, and the posting of warning signs.

As for differences between the two countries, the Swedish law allows the banning of chemicals in the workplace. OSHA is not allowed to do this. The Swedish agency may also ban or forbid certain workers from working with a chemical if they are particularly susceptible. OSHA cannot do this; it has said that if a woman is taken out of a job because of exposure to a substance that may cause birth defects, she is entitled to the same pay level. These are the controversial "rate retention provisions."

Moreover, the Swedish statute allows the regulatory agency to allow use of a certain chemical only after special permission has been granted. OSHA considered a "use permit" system, but this was turned down. Finally, the Swedish statute states that the agency has the right to require workers to be given information about the chemical composition of substances with which they work. OSHA doesn't have this power, although the agency has a proposed regulation dealing with this right.

Dr. Kelman said the Swedish system relies extensively on local safety stewards and committees in the workplace. The law and collective bargaining agreements require establishment of labour/management safety committees with a one-person labour majority. There is no similar provision in U.S. law.

The Swedish statute gives individual workers or safety stewards the right to stop work in an "imminent hazard" situation. This applies mostly to immediate and serious safety hazards. "It does not apply to a long-term exposure to a substance," Dr. Kelman said.

The final difference he cited between the two countries had to do with enforcement. The United States is the only country where the occupational safety and health enforcement system is based on the rather controversial concept of "first instance sanctions"; that is, "the regulations have the immediate force of law, and it's expected that the employer has complied with them before the inspector comes to the workplace. Therefore, the inspector has a right when he comes in, if you're in violation of the regulation, to fine you immediately." He said this is normally what happens, although "the fines are not enormous."

In contrast, the process in Sweden would be longer and the employer would not be fined the first time around. Moreover, "The Swedes place a lot of reliance on the local safety organization....to the inspectors who are there all the time, so to speak...to see to it at the local level that regulations are complied with."

Dr. Kelman then moved on to the content of implementing regulations and the process of developing regulations. Until recently, the content of regulations in the two countries has been "surprisingly similar, in general." Decision-makers have chosen to adopt more rather than less protective alternatives to safeguard workers and in general have opted for engineering controls rather than personal protective equipment.

"What's very different is the process of developing regulations in the two countries," he said. The process in the U.S. system is "probably unique" in terms of length and complexity; in terms of the amount of data gathered, particularly on feasibility and cost; and in terms of the involvement of lawyers and the courts.

He then gave a brief, simplified description of OSHA's procedures in developing a new regulation: preparation of a criteria document giving the medically-based threshold limit value; publication in the Federal Register of an "Advanced Notice of Proposed Rulemaking" inviting comment, along with a preliminary review of costs and benefits; publication (after receiving comments) of the proposed rule; the holding of quasi-judicial public hearings; issuance, in conjunction with promulgating the rule, of a statement of reasons, stating why they did what they did and why they didn't do what some people asked them to do; issuance of an environmental impact statement, a regulatory analysis (costs, benefits and inflationary impact) and -- a new requirement -- a discussion of differential compliance methods for small and large businesses, so there is not an excessive impact on small business.

"After all this has happened, then people can challenge the regulations in the courts," Dr. Kelman said. In fact, virtually every OSHA regulation has been challenged in court; some as far as the Supreme Court.

In Sweden and most other countries, the system is "infinitely easier." Unlike the United States, there is no statutory requirement to go through these steps. The Worker Protection Board gets a committee together representing labour, management, and the government. "They sit down around the table, meet for a few months, and decide what they want to do, and then it comes out....No lawyers, no cross-examination..." He said this process

does help to obtain agreement between labour and management on the content of regulations, to a degree not achieved in the United States. The U.S. business community "has essentially opposed everything OSHA has done." Initially, labour thought OSHA wasn't doing enough, but recently has become pro-OSHA.

Dr. Kelman then turned to regulations of hazardous substances outside the workplace. The key U.S. agency here is the Environmental Protection Agency; it was set up in 1970 and has a reputation as an aggressive agency. The EPA enforces the Clean Air Act of 1970 which, among other things, deals with hazardous air pollutants. The Act allows the EPA to establish emission standards for given sources and ambient air standards for the overall level of air quality. The Act's amendments are up for re-authorization in Congress this year.

There is also the 1972 Water Pollution Control Act, which includes provisions for emission standards for hazardous water pollutants such as asbestos; and the Safe Drinking Water Act, which allows establishment of threshold limit values for the presence of various hazardous substances in drinking water. The 1976 Resource Conservation and Recovery Act deals with the disposal of hazard waste. (Dr. Kelman said there is a general fear in the United States that hazardous wastes are like a "time bomb.")

Finally, there is the Toxic Substances Control Act of 1976, known as "TOSCA." Dr. Kelman described it as the most important act dealing with hazardous substances and said it has four types of provisions most applicable to substances like asbestos:

(a) It authorizes the EPA to set up the first general listing for all chemicals in use in the United States.

(b) It authorizes the EPA, if it wishes, to require retrospective testing of chemicals already in use, where it is felt there is not enough information currently available in the scientific literature about chemicals that may be hazardous.

(c) It requires companies to notify the EPA when they plan to put a new chemical on the marketplace, and it allows EPA to require pre-market testing.

(d) It allows the EPA to restrict production or use chemicals deemed hazardous. These restrictions can take the form of labelling, exposure limits, production limits, or a complete ban.

(There is a similar framework in the Swedish Product Control Act of 1973, which has many identical provisions.)

Dr. Kelman then reviewed how the framework for legislation and regulation applies to asbestos. The asbestos regulation in the workplace was one of the first OSHA developed; it was the only one challenged in court by labour on the grounds that it was not strict enough. He said there is increasing concern about the contribution of asbestos exposure

to occupational cancer -- especially because there seems to be "a genuine occupational health epidemic" among shipyard workers exposed to asbestos during World War II. (In a remark that was to elicit much comment during the question period, Dr. Kelman mentioned the death from mesothelioma of actor Steve McQueen, who had been a shipyard worker during the war.)

With this increasing concern, OSHA has proposed making threshold limit values stricter. In 1975, they proposed a .5 fibres per cc regulation; Dr. Kelman said they were planning to lower that to .1 fibre per cc, but was uncertain whether OSHA would get this out before the end of the Carter Administration.

In Sweden, in 1974, the asbestos regulations were made tougher, and there were widescale bans of asbestos in the workplace. It has been banned in paints, glues, floor and wall coverings and, most controversially, in cement (except for asbestos cement piping) for use in the workplace. It is still allowed in brake linings, in come protective equipment, and in some specialized uses. The goal is to gradually eliminate asbestos from the workplace. Dr. Kelman said this has been almost the only use of the power to ban hazardous substances in Sweden.

With respect to general environmental levels of asbestos outside the workplace, there have been no threshold limit values proposed in the United States under any of the acts except the Clean Air Act and "TOSCA" ; the former does set emission standards which, in part, ban spraying asbestos in buildings. "But the general industrial standard is a weak one, that requires no visible emissions of asbestos....If you get to the level where asbestos emissions are visible, that's very, very high. So it's not a particularly strict standard."

However, the EPA has been devoting much attention to asbestos. The agency has proposed a rule requiring school inspections and had been planning to propose a second stage requiring schools to clean up. Congress had passed an act making grant money available to schools wanting to clean up. Dr. Kelman said it is unclear what will happen to this program under the Reagan Administration.

Recently, the EPA also announced it was considering selected bans of asbestos for various uses and the possibility of a total limit on the amount of asbestos that can be imported or used in the United States. This is a "market oriented" approach. Here, again, the fate of the program under the new Administration is unclear.

Dr. Kelman concluded by discussing the growth of opposition to regulation, in the United States in particular. Much of it is psychological; the growth of regulation was rapid in the 1970's, and many business people, who were not used to government control, "suddenly perceived that a lot of their lives were being taken up by government coming in and saying, "your've got to do this, you've got to do that." This not only rankled psychologically, but many managers felt that coping with regulations was taking too much time.

In addition, there is a tradition in the United States of hostility to government. And finally, there is the poor economic environment, which many in business try to blame on excessive government regulation. This has resulted in "a dramatic slow-down in the number of new laws that have been passed," and increasing attention to the costs of regulation. Moreover, there have been attempts to centralize the process -- to give the President more control and the agencies less freedom to do what they want.

"The big question is what's going to happen under the new Reagan Administration, given Governor Reagan's hostility towards a lot of the environmental and safety and health regulations." Dr. Kelman said it has been proposed that a one-year moratorium on all new regulations be instituted, to be followed by a "trade-off" situation, whereby every time an agency issued a new regulation costing so many millions of dollars, they would have to rescind an earlier regulation of equivalent economic value. "So the fate of many of these asbestos regulations...is very much uncertain."

Regarding public attitudes, Dr. Kelman said there's no doubt that the idea of "getting government off our backs" evokes a public response. But, at the same time, public opinion surveys indicate that this general anti-government attitude doesn't translate into hostility towards health and safety regulations. He said advanced Western nations place too great a value on human health to simply shunt aside measures to protect that health, despite short-term policital trends. "I suspect, longer-term, this kind of regulation is going to continue to be around, continue to be strict."

D.2 Address by Dr. G. Bruce Doern, Director, School of Public Administration, Carleton University, on "Frameworks for the Regulation of Hazardous Substances in Canada"

Dr. Dewees next introduced Dr. G. Bruce Doern, who is the Director of the School of Public Administration, Carleton University, Ottawa, Ontario. He is the author or editor of several books and articles on Canadian public policy and public management, including Science and Politics in Canada (McGill-Queen's Press, 1972); The Politics and Management of Canadian Economics Policy (co-authored with R.W. Phidd) (Macmillan of Canada, 1978); The Regulatory Process in Canada (Macmillan of Canada, 1978); Government Intervention in the Canadian Nuclear Industry (Institute for Research on Public Policy, 1980); and Public Policy in Canada: Organization, Process and Management (Macmillan of Canada, 1979).

Dr. Doern's address to the second public meeting of the Royal Commission on Asbestos centred on "Frameworks for the Regulation of Hazardous Substances in Canada."

Dr. Doern started by pointing out that this was his "fourth hazardous royal commission" -- which, he said, points out that Canada has tended to handle these problems on a "hazard-by-hazard basis." And yet, it is very clear that the terms of reference of the Royal Commission on Asbestos, and simply the political and economic reailty in which it must function, will have to address the more general phenomena of hazardous substances and how they are regulated.

Dr. Doern said it is first essential to appreciate the physical realities and production processes by which hazardous substances are generated: as creation, manufacture, distribution, transportation, marketing, and the disposal of waste products from all of the previous processes. This means there are many points at which governments can choose to intervene or not intervene, a fact relayed by the "parade of statutes" in most industrialized countries.

Dr. Doern described Canada's Environmental Contaminants Act as a "much, much meeker, milder, timid version" of the U.S. "TOSCA". He noted that the Canadian constitution gives provincial governments much greater powers to regulate the workplace than it does the federal government.

Governments have chosen to intervene, not only on a hazard-by-hazard basis, although many recent initiatives, including the parade of royal commissions, are triggered by concerns about particular hazards. This raises questions about why there is a political and media response to "issues like asbestos on the one hand and quite different responses to things like auto safety on the other. And yet, in certain ways, they are part of a large class of problems."

Dr. Doern added that governments have a limited number of ways to influence or change behavior. They can exhort: that is, use educational programmes to seek to persuade people to change their behavior. They can use guidelines to coax workers, consumers, and industries (some of which are publicly-owned, which puts the government in the position of regulating itself). Governments can offer incentives -- in part, an alternative or complement to direct regulation. He noted that companies that might be the object of regulations might, at the same time, be receiving economic incentives from other government departments. Finally, there is regulation itself, best seen, politically, as "the development of rules of behavior backed up by the sanctions of the state."

Dr. Doern then went on to describe economic, political, and administrative frameworks and illustrated each type.

He said Canada has clearly had its own version of the regulatory reform movement referred to by Dr. Kelman, although it has not been as strong or symbolic as that in the United States. "It arises from sheer economic difficulties we're experiencing -- a fairly frantic look for anything and everything that somehow can reduce inflation, improve productivity and so on." There have been a number of attempts within governments to assess regulation in a more intensive way.

This has also been reflected in an economic concern about the degree to which the "first generation" of regulatory reform in the early 1970's was "perhaps too enamored of capital intensive solutions"; that is, the notion that basic production processes in the workplace should be reformed rather than looking for "softer," less capital-intensive methods (for example, wearing a mask).

The impetus for reforming the regulatory process through economic analysis has been reflected by the fact that the Treasury Board now expects, as a guideline requirement, that a social and economic impact assessment will accompany a proposed regulation. If an agency is contemplating a new regulation and determines that its cost to the private sector will exceed $10-million, then that agency will be required to go through a process and publication routine somewhat like the U.S. situation.

Dr. Doern described this as the most formal expression of concern "that what we need in regulatory reform in a climate of economic uncertainty is second sober thoughts" about whether any regulation should occur at all and whether regulation is the best way to go, or whether economic incentives and educational programmes, "or some combination of the carrot and the stick" would be the best thing to do.

He expressed the personal view that the entire area of occupational health and safety and of hazardous substances, "given the current reality of political priorities in this country...is very clearly on the fringes of political activity. One would not gauge that from the media, but in terms of the reality of economic and political power, it is on the fringes. It is not a high priority item, the existence of royal commissions to the contrary notwithstanding."

Turning to political factors, Dr. Doern said that the role of the media is intrinsically important and not to be politically underestimated. Workers and communities often learn first about a hazard through the media. He added that the Canadian system is "far less open" than the U.S. system. There have been some improvements in recent years, but regulators here are still "almost frightened to death of emulating the American system."

Another factor, which he identified as important in the area of administration and compliance, is the "reasonably consistent" position take by unions concerning standards. "Unions in Canada and elsewhere have a very strong preference for fixed, firm standards. They want rules of behavior. They want them partly for reasons of distrust of both management and regulators. They want them in case legal confirmation of standards and blame and guilt might have to be determined. They want them so that, in principle, there will be some notion of equality of treatment in law. But above all, they want some certainty; this despite a reasonable awareness of the scientific problems we have talked about."

This raises a conflict between two good things: equality before the law and justice, which may require different people to be treated differently. (For example, a small business might argue that it should not have to meet the same standards as a large business.) "So we want both. We want uniform standards because we want certainty, predictability, equality before the law. We want justice, which means we want...special circumstances to be taken into account." Thus, it is difficult to reach accomodation that is acceptable to all parties all the time.

A further political issue is what Dr. Doern referred to as "the politics of science." Alluding to the "sense of caution about causality"

demonstrated earlier in the day, he said such caution is "absolutely essential" in science. He made a distinction between scientific evidence and political evidence. He says that at first in these "single hazard" situations only a handful of people or "cases" are brought forward. "But they are real; they are a form of evidence; they are the kind of thing that the media quite properly pay attention to."

The other form of evidence comes from science, which must be more cautious and cannot afford to treat single cases as evidence; which must assemble a range of information about the phenomena. Therefore, the scientific community has both a "desirable vested interest" and simply a "vested interest" in more research; unions want action instantly on single cases, single hazards, without necessarily taking into account the impact of these choices on other "hazards, in which there's less certain knowledge, or which may simply affect other people who are not their concern."

Regarding compliance, Dr. Doern said it is hoped that "things will in fact change; that it will somehow be better. It's important to ask questions about what is real compliance, what is real change and improvement." This raises a number of issues. For example, several provinces (but not the federal government) have moved to consolidate inspectorates in labour departments; the intention is that these people, who were formerly scattered, will look upon the workplace as such as their purview. This has been done with various degrees of enthusiasm.

Another factor bearing on whether compliance is real or bogus relates to inter-agency dependence and intergovernmental dependence. Often, adding yet another hazard to be regulated simply means adding another set of duties to the workload of existing inspectors, often working for departments other than the one that has launched the new regulation. This raises questions about how much time and knowledge the inspectors have.

Looking at inspectors' duties, it appears they would have to be "almost bureaucratic supermen" who have quite sophisticated knowledge about a wide range of hazards. Yet, there is concern about excessive bureaucracy. "We have an apparent wish to have enforcement but have no enforcers... We don't want too many inspectors roaming around our shops, but somehow we want good inspection."

Dr. Doern concluded that the Commission could not look at asbestos on its own, but has to look at it across the entire production cycle and in relation to a number of other hazards, at least in terms of public policy. Finally, he said that "politics is not a series of choices between goods and bads; it's more often a choice among goods. We have encased in this system of regulation of hazardous substances several goods, not too many bads, and series of problems in the choice of principles, some of which conflict with others."

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Following the presentations by Drs. Doern and Kelman, there was a question period.

Mr. Lionel Piuze, a representative of the Quebec Asbestos Mining Association, said Dr. Doern's presentation finds the support of the mining industry and his comments are "well-received by both sides of the fence" (a reference to the industry and unions). He said he could not say the same thing about Dr. Kelman's talk and said he was shocked by the reference to the claim that Steve McQueen died of an asbestos-related disease. He said that McQueen (and John Wayne) had done many movies in states where there had been numerous nuclear tests. "You forget about that; you jump immediately on asbestos. I am one of those who cannot stomach that kind of medicine, especially from public servants."

He added that there have been serious fires in California that have destroyed many homes near Los Angeles. "The only ones that remained intact were the ones that were covered with asbestos material." He said that asbestos is not all bad and, as someone who has worked in the asbestos industry for more than 40 years, "I respect it and I'll fight for it."

Dr. Kelman responded by noting that he was not an expert on asbestos and "I lay no special claim to knowledge of the epidemiology or toxicology of asbestos." Nevertheless, he said, it is his understanding that there is considerable evidence linking mesothelioma -- the cancer from which Steve McQueen died -- to asbestos exposure. This is "fairly well-accepted among epidemiologists who are experts in the area, which I do not claim to be." He said it's also a fact that McQueen was a shipyard worker exposed to asbestos.

The moderator, Dr. Donald Dewees, pointed out that this was not the proper panel to be discussing questions of causation, which had been the subject of the morning session.

Dr. Michel Lesage, a medical consultant with the Quebec Asbestos Mining Association, agreed with this comment and said this is why he was suprised to hear Dr. Kelman make the reference to Mr. McQueen. He also challenged Dr. Kelman's comment concerning the occupational health epidemic among shipyard workers, saying he did not quote studies which concluded "there is no industrial disease epidemic in the United States at this moment." He said he hoped that people who come to the Commission "will have scientific statements and if they want to show something on one side, they will say also the things for the other side."

Dr. Kelman said he was not giving his personal view on asbestos exposure. His point was that the various reports cited were reasons for the great amount of attention given by U.S. authorities to asbestos -- the only chemical subjected to more than one rulemaking by OSHA. "I am not competent to endorse or not to endorse those reports. I am not saying these reports are not true; neither am I saying they're true." Dr. Kelman stated that these reports, like the McQueen incident, whether or not they are true, help to explain the intensity of public concern over asbestos in the U.S.

Ms. Christine Mitchell, a Toronto lawyer, asked Dr. Doern to clarify his comments on the trade union approach to regulation.

Dr. Doern said this was based on statements to other royal commissions and in public hearings, where the unions on the whole have expressed a preference for the establishment of minimum standards of regulations, on the assumption that there is some threshold limit value. (This has not been the case in Saskatchewan, which has "preferred to leave it as a guideline and then to achieve the lowest possible exposure through other devices. They've preferred not to establish a firm standard.")

He said this attitude on the part of unions may stem from the "sense of distrust that many of them have, quite properly, developed over a period of time about whether they have been able to trust the information; whether they've been able to get the regulators to inform them of developments." So their position during earlier commissions in 1978/79 was to demand firm standards; they didn't want it left flexible because this was too easy a way out.

Ms. Mitchell commented that she was confused by Dr. Doern's comment that unions were insisting on a hazard-by-hazard approach and the fact that this might have an adverse effect on other regulations.

Dr. Doern said he was not referring to unions saying there should only be a regulation on asbestos. "They've pressed for occupational health bills as a whole in virtually every jurisdiction in Canada, on the assumption that there are a lot of problems of this kind and they should be addressed across the board." He said the comment Ms. Mitchell was referring to was a reference to "the broader way in which this issue has come on the agenda in Canada." For example, it was essentially political and media criticism of the conditions faced by uranium miners in Ontario that launched the Ham Commission of 1976 (i.e. The Royal Commission on the Health and Safety of Workers in Mines). "But the unions' position has been to look for overriding legislation. In fairness, it's also important to say that Canadian unions...have not pressed these claims on occupational health until very recently, that is, the last four or five years."

Mr. Eddy Cauchi asked Dr. Kelman if it was true that Swedish workers must be told about being exposed to any hazardous material, while this is not the case in North America.

Dr. Kelman said the Swedish law does state that workers have the right to be informed about materials they are being exposed to, but he was uncertain whether implementing regulations had been developed. In the United States, there are regulations covering different specific hazardous substances such as asbestos and others requiring new workers to be given information.

Mr. Cauchi said he and his fellow workers and the widows in the audience were "sick to our stomach" because of certain actions taken by Johns-Manville in the past. He referred to a case in 1933, when the company Board of Directors settled 11 workers' lawsuits for $35,000 on condition that the individuals not bring any more suits. Then he quoted

from what he described as a confidential 1949 report by Dr. Smith, then the company physician, saying that company workers should not be informed of their illness because they would become unhappy. The attitude, Mr. Cauchi said, was that "if you don't tell them, they're going to come back to work and be happy ever after. Now I wonder who is sick to his stomach in here and who is being on one side?"

The session was concluded with an impassioned statement from Mrs. Odette Dodds, the widow of asbestos worker John Dodds, who died in 1978. "I am a woman with great determination and have a mind of my own....My husband worked for Johns-Manville company for 23 years. He'd never been sick a day of his life, never missed a day of work. In 1974, my husband was forced to retire due to industrial diseases. In 1977, my husband became a human guinea pig, to find out before he died how much asbestos could damage a human body.... I saw my husband going through hell. He (donated) his body to anatomical and medical research. The hardest part for us was to...find a doctor we could trust and we found three of them."

When her husband died "an autopsy was performed. His body sections were preserved and here's what we found: 100% asbestosis, silicosis, severe fibrosis on both lungs, thyroid malignant carcinoma, one vocal chord paralysed, spinal damage due to radiation treatment. He received 100 radiation treatments in 12 and a half days, which destroys the cancer and also the evidence."

"Cancer started on his back -- melanoma -- and I saw that cancer grow right on the top of my husband's spine and it changed four different colours and it started to grow just like a mop and we couldn't stop it. He had scirrosis of the liver, he lost both legs, he lost his hands, his arms, became deaf and he couldn't speak any more. And the more I think about it, the more I want to do something about it."

"In 1955/56, we almost all ended up in our own graves through Johns-Manville negligence. It took my husband and I many years to find out about asbestos and when we found out about asbestos, the magic mineral with dust that kills, it was already too late, because no matter how strong you are or how healthy you are, there is no escape for anyone."

"If I may say so, we should have had this kind of public meeting a long time ago, because if we had, I don't think we'd be in the mess we are today. Is it through progress, industry, negligence, greediness, carelessness, stupidity, ignorance, fear, panic -- your guess is as good as mine."

"One of the biggest problems is that we don't like to think and talk about the truth. Three quarters of the time we are our own worst enemy, and it is about time that we realized the facts and changed the law if we want to keep this country healthy and prosperous. The thing I want the most is not here anymore...and I can't do a bloody thing about it. That is the Canadian law. You know and I know it's not right."

Mrs. Dodds said she had a friend with her whose husband died after working for Johns-Manville for 30 years. His death was "not compensable," so her friend had to go back to work. "I know it's depressing, but I don't try to escape -- I just try to prevent another disaster in 20 to 30 years from now, through my own experience and tragedy."

She said she'd immigrated to Canada in 1953 and "I think the world of my new country and even if I have to fight this until the day I die, I am willing to do so. When I lost my husband, I lost everything; there is no more future for me. I've got children and grandchildren; I'd like to see my loved ones and others have a future....I worked for 21 years and I lost everything through Johns-Manville's negligence and that's not right, that's not fair."

She said that when her husband started with Johns-Manville in 1952, "conditions were so bad that the workers couldn't see each other."

Mrs. Dodd's concluded her statement by saying she had always supported her husband 100% and always would. "Rest in peace, John. I shall live on with pride and dignity. I shall carry your good work....I shall stick to my guns till the end...John, I'll never forget you -- I won't forget, nor forgive, nor give up. Giving up is failure; I don't want to be a failure -- I want to be a winner. I'll be back each time here."

Commission Chairman Dr. Dupré told Mrs. Dodds that she will always be welcome.

The meeting was adjourned.

APPENDICES

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APPENDIX A

ADDRESS BY:

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THE PROBLEMS OF MEASUREMENT OF ASBESTOS

Prepared for the Royal Commission on Asbestos
Second Public Meeting, December 12, 1980

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1. INTRODUCTION

1.1 Varieties of Asbestos

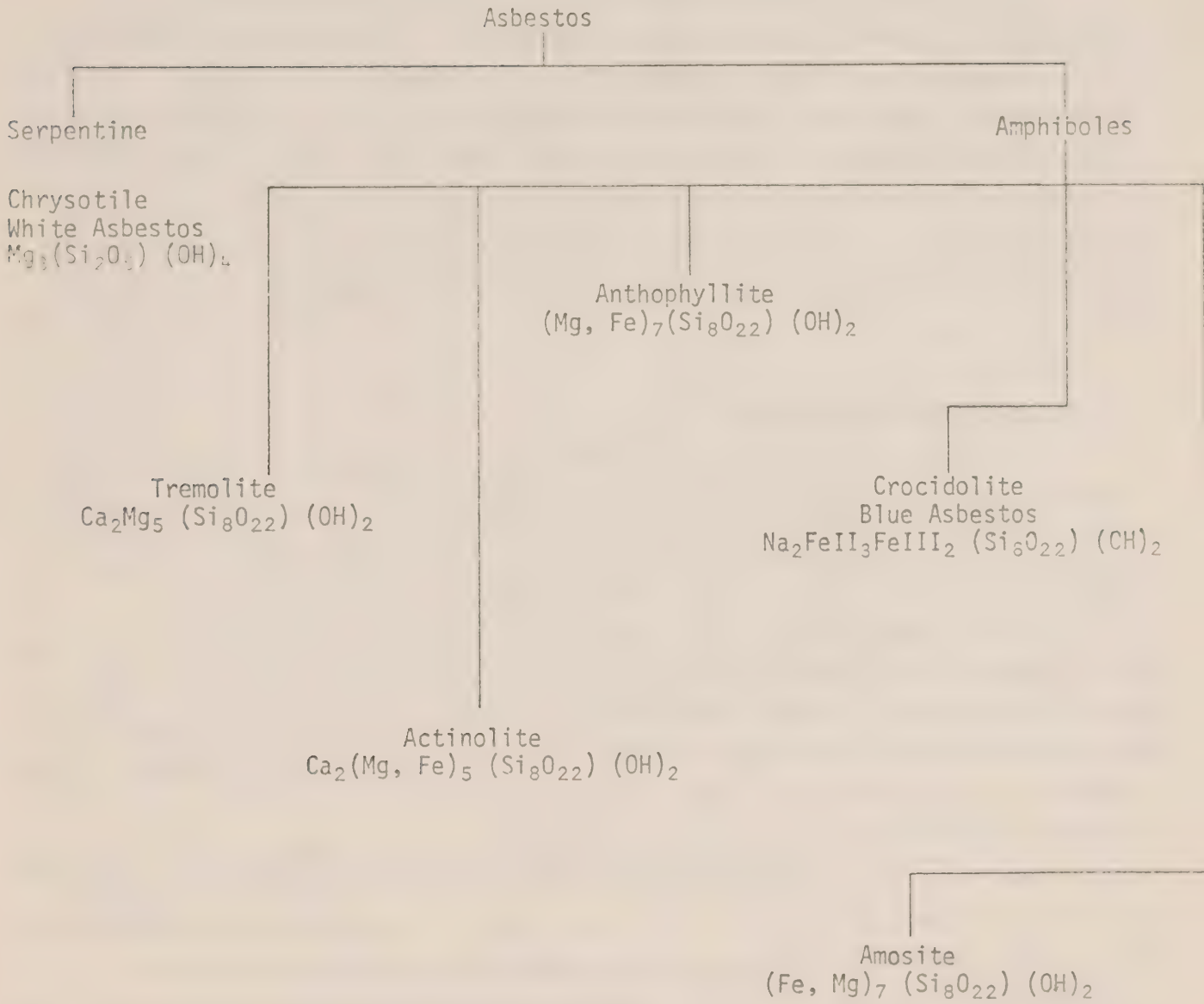
Asbestos is a term used to describe a number of minerals which have the property that they can be separated into long silky fibres. The three main kinds of asbestos which have found commercial use are chrysotile, amosite and crocidolite. Of these, chrysotile is by far the most abundant and the most used. Other less important varieties are anthophyllite, tremolite and actinolite. Figure 1 shows the compositions of these types of asbestos. Chrysotile is the only member belonging to the serpentine group of minerals; all of the others belong to the amphibole group. Amosite is not in fact a mineral name but is an acronym for Asbestos Mines of South Africa. Amosite usually consists of the amphibole mineral grunerite, but cummingtonite and tremolite in variable proportions are often present in some specimens. It will be noticed in Figure 1 that several of the minerals exhibit variable composition within the same species, and such variations may even occur from one fibre to the next in material from the same deposit. All of the minerals may also incorporate small concentrations of other elements, examples being crocidolite and amosite which often contain small concentrations of calcium and aluminum. Since no single element is uniquely associated with these minerals, it is not possible to detect or identify small quantities by simple chemical analytical methods, if other silicate minerals are also present.

1.2 Asbestos and Non-Asbestos Varieties

The asbestoses all have non-fibrous polymorphs of similar composition. The asbestos variety is composed of particles which are extremely thin and have very high length to width ratios. In general, these fibres are also very flexible, whereas particles of the non-asbestos variety usually cleave into needle shaped fragments which do not display the property of flexibility. The difference, therefore, between the asbestos and non-asbestos varieties of the

Figure 1

Principal Varieties of Asbestos



same mineral is concerned with a mineralogical property known as *crystal habit*. Although in a large sample it is relatively simple to discriminate between the crystal habits of the fibrous and non-fibrous versions of the same mineral, this is not true when a single small particle of the mineral is examined in a microscope.[1]

1.3 Definition of "Fibre"

A "fibre" is almost universally defined as a particle which has a length to width ratio (aspect ratio) exceeding 3:1. This fibre definition is strictly arbitrary and has no biological or mineralogical significance. Unfortunately, many other common minerals when ground yield elongated fragments which can be classified as fibres under this definition.[2] The 3:1 fibre definition has very serious consequences in electron microscope analyses for asbestos, and an increase to 10:1 or greater has been suggested[2,3] as a means of reliably discriminating between asbestos fibres and elongated fragments of non-asbestos minerals.

1.4 Use of the Term "Asbestos"

It would be attractive to confine the use of the term asbestos to the minerals which are commercially exploited. However, this could be misleading since many mining operations which do not primarily produce asbestos have minerals in the ore body which are indistinguishable from asbestos and are of unknown health hazard. For example, the minerals tremolite and actinolite occur in some deposits of vermiculite, and anthophyllite has been found in some talcs. It is a controversial matter whether such particles of amphibole found in some of these products should be considered as asbestos, since amphibole is one of the more common minerals in the earth's crust.[4] It is only amphibole crystallized in the asbestiform habit, which is comparatively rare, about which there is a substantial amount of health data available. A precise definition is important for the workplace situation since this definition controls whether the individuals working in such an operation should be considered to be asbestos workers or not. A number of other problems arise when environmental measurements are made in the vicinity of a mining or processing operation. It is then necessary to discriminate between fibres which may be discharged from the

process into the environment and other particles of an elongated or fibrous nature which may already be present.

1.5 Considerations of Fibre Size

The medical community is currently able to give only an indication of what should be measured in the case of asbestos fibres. Work by Stanton[5] and later by Pott[6] indicates that fibres less than about 0.2 µm in diameter are considered to be the most significant in carcinogenic activity (Figure 2). Moreover, examination of the fibres found in the lungs of mesothelioma patients at autopsy indicates that the majority were below that diameter and that only a small proportion had lengths exceeding 10 micrometres (µm).[7] In general, by far the greatest number of asbestos fibres found in both the environment and the workplace are below the resolving power of the optical microscope.[8] In the workplace atmosphere, control measures have been established using simple phase contrast optical microscopy. It is, however, important to recognize the limitations of this approach: the measurement is non-specific, in that all particles having aspect ratios exceeding 3:1, rather than just *asbestos* fibres, are counted, and the resolution is inadequate to detect fibres having diameters less than 0.2 µm whatever their lengths may be. In view of the current uncertainty about the biologically relevant parameters of asbestos fibres, wherever possible the safest course would be to measure as many of the physical and chemical parameters as possible in every sample. Where a situation is already well-characterized, such as the asbestos workplace, effective dust control measures can be implemented using rather more simple measurement criteria.

2. MEASUREMENT OF AIRBORNE ASBESTOS IN WORKPLACE ATMOSPHERES

2.1 Definition of "Fibre"

In the asbestos-using workplace it can be assumed that any airborne fibres are likely to be asbestos of the variety in use. Accordingly, the fibre is defined in an arbitrary way as a particle having an aspect ratio greater than 3:1, longer than 5 µm and less than 3 µm in diameter. This arbitrary definition allows the use of the membrane filter method, which is a simple and inexpensive technique to measure airborne fibre concentrations.

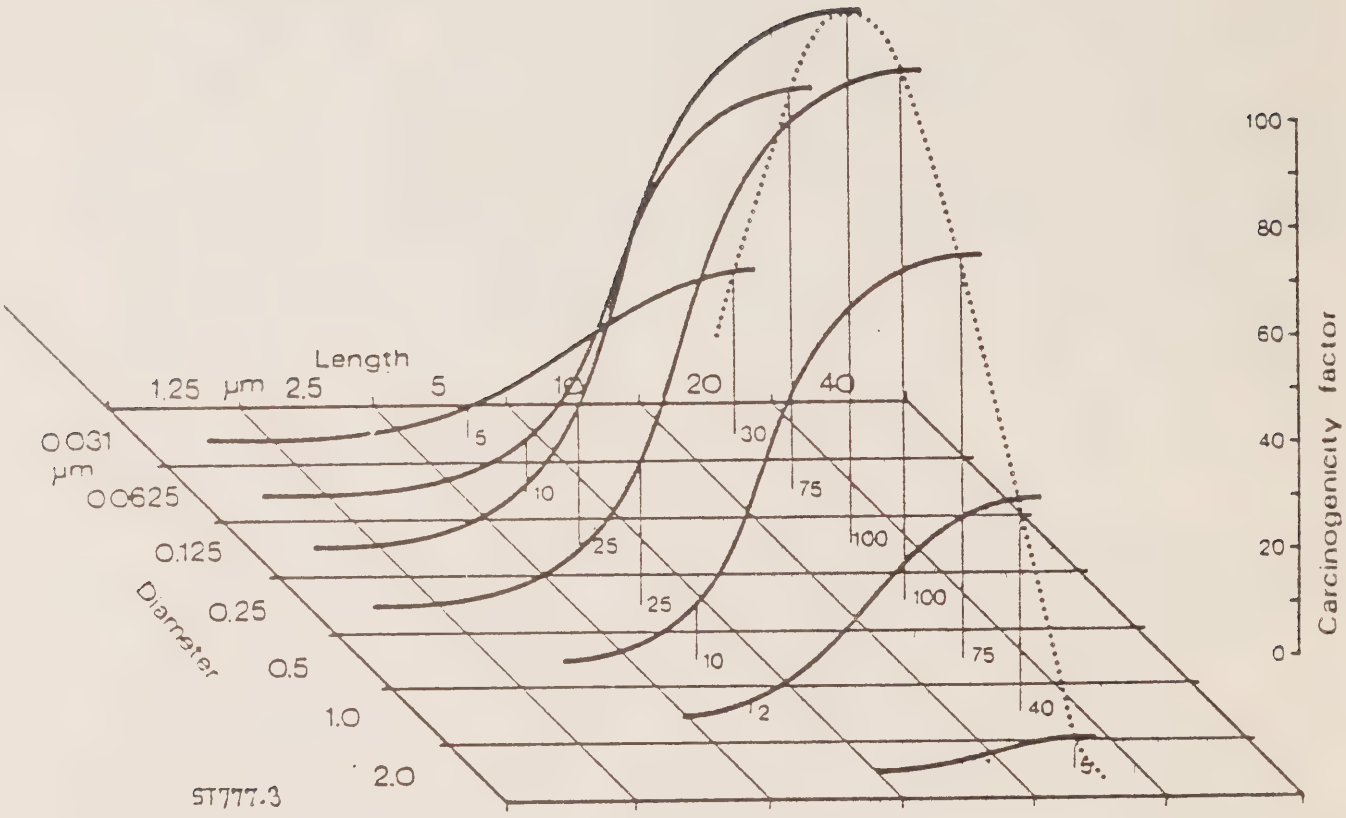


Figure 2. Hypothesis on the carcinogenic potency of a fibre as a function of its size with some data on "carcinogenicity factors", (After Pott (1978)).

2.2 Membrane Filter Methods

Airborne dust samples are collected on membrane filters, which are then examined by transmitted light microscopy after the filter itself has been rendered transparent. This is achieved by immersing the filter in a liquid of the correct refractive index. However, when this is done, it is found that under normal bright field illumination, the fibres are almost invisible, since they have indices of refraction very close to that of the cleared filter. Phase contrast illumination is capable of revealing objects which have refractive indices only slightly different from that of the medium in which they are immersed. Phase contrast illumination is used to evaluate the filters at a magnification of about 500. It is most important that the optical microscope be correctly aligned as failure to do so may result in counts reduced by up to 50%.

Several methods of clearing the filter are available. The mounting solution used in the NIOSH (National Institute for Occupational Safety and Health) method[9] is a 1:1 mixture of dimethyl phthalate and diethyl oxalate with incorporation of about 0.05 grams of dissolved membrane filter material per millilitre of solution. Unfortunately, this mounting medium itself generates "fibres" during storage, and extreme care has to be taken to ensure that the mounting medium is fresh. Microscope preparations using this technique are not stable, and after about 24 hours the slide is of no value. A technique recently published by the AIA (Asbestos International Association)[10] uses a different mounting technique which results in a permanent slide that can be stored for future reference. In this method, acetone vapour is used to clear the filter, producing samples on which the fibres tend to lie more in one plane than is the case with other methods. Essentially, the filter is exposed to a jet of acetone vapour produced by a flask of boiling acetone. The filter then sets to a clear transparent film, and a drop of glycerol triacetate is added, followed by the cover glass.

2.3 Inter-Laboratory Studies Using the Membrane Filter Methods

Beckett and Attfield[11] reported large inter-laboratory differences when identical samples were counted by phase contrast microscopy. There were significant differences between the results reported by novice counters and experienced counters. The main causes of the differences were found to be uncertainties in counting of irregular aggregates of fibres, misalignment of the microscope, and failure to scan the full depth of focus in which fibres were present. Unfortunately, inadequacies in experience, operator fatigue and microscope misalignment all cause a low result to be reported. It is clear that optical fibre counting of workplace atmosphere samples should always be accompanied by a stringent analytical quality control program.

It has been shown that, although a single laboratory can achieve good reproducibility, inter-laboratory analyses show greater variability.[12] This is demonstrated by an inter-laboratory fibre counting exercise which was performed in 1978 using the NIOSH technique. The eighteen individuals involved in this study were all experienced in phase contrast fibre counting. Some of the results are shown in Table 1. It can be seen that there was a significant range in the counts reported for each filter. In one case, the ratio of the highest to the lowest count was a factor of 6.2. On this filter sample, one counter was able to find 6.2 times more fibres than another counter.

It has been found that significant discrepancies exist between operators in the way in which specific arrangements of fibres are counted. For example, split fibres or fibres with particles attached should be counted according to constant criteria. However, no suitable standard exists and the most common rule when clumps of fibres are encountered is to move to another field of view in cases of difficulty. Such a rule automatically introduces a low bias into the value, since the most heavily loaded fields are disregarded. The AIA method introduces the concept of diagrams of specific fibre arrangements with defined methods of enumeration, as an attempt to reduce this variability.

TABLE 1

INTER-LABORATORY NIOSH FIBRE COUNTING OF FOUR FILTERS (1978)

(Values in fibres/mL assuming four hour sampling at 2 litres/min. Fibre Counts made by 18 experienced counters)

	ASBESTOS OPERATION			
	Carding	Twisting	Milling	Milling
Mean Value	1.36	1.00	0.99	0.64
Lowest	0.63	0.51	0.33	0.41
Highest	3.04	1.43	2.04	1.32
Range (High/Low)	4.8	2.8	6.2	3.2

The AIA method incorporates the use of resolution standards so that the performance of the optical microscope can be standardized. Moreover, the permanent nature of the final sample prepared by the AIA method permits exchange of slides between laboratories for inter-laboratory checks to be made.

2.4 Suitability of Optical Fibre Counting for Exposure Assessment

The validity of the membrane filter method for exposure assessment is still questionable and must be considered carefully.[13] It has been demonstrated that the dimensions of fibres and their size distributions can vary significantly with fibre type and the particular process being monitored. Thus the number of fibres observed in the phase contrast microscope may comprise different proportions of the airborne asbestos fibres to which persons in different industries are exposed. In many airborne distributions, the phase contrast optical microscope measurement records only 2% - 25% of the fibres longer than 5 μm, and perhaps only 0.1% - 1% of the total number of fibres.[14] It is not known whether the fibres observed in the optical microscope are the ones responsible for the health effects, and it may indeed turn out that the index of exposure is not related to this measurement at all.

It has also been demonstrated that the detection level of the phase contrast method is about 0.1 fibre/mL.[12] This is very close to the proposed maximum level of 0.2 fibre/mL for crocidolite.

2.5 Limitations of Phase Contrast Fibre Counting Methods

It should be recognized that the phase contrast technique yields a *fibre* count only, and is not specific for *asbestos* fibres. The use of this method, therefore, has limitations when applied to samples containing acicular particles, for example, talc or gypsum, and *should not be implemented without a full qualitative understanding of the sample.* Such a qualitative understanding can be obtained using polarized light microscopy or electron microscopy.

An example of inappropriate use of the phase contrast technique can be found in air sampling conducted in a vermiculite exfoliation plant. The

particular vermiculite in use had been demonstrated by electron microscopy to contain no detectable asbestos fibres. Table 2 shows a comparison of the results obtained for the air samples, by both phase contrast fibre counting and electron microscopy. It can be seen that a remarkably high fibre count was obtained in a location where no asbestos was in use.

The U.S. National Bureau of Standards also reports that the technique is unsatisfactory in the presence of talc. Plates of talc viewed on edge can be classified as fibres. The phase contrast count would also include wollastonite, glass fibre and other materials such as cellulose.[15]

3. MEASUREMENT OF ASBESTOS IN THE GENERAL ENVIRONMENT

3.1 Unsuitability of Optical Methods for Environmental Measurements

In the environment it is unusual to encounter the large fibre bundles which are found in the asbestos workplace atmosphere. Any asbestos in the general environment is usually present as small diameter fibres which are not detectable by optical microscopy on account of the limited resolution. Since no attempt is made to discriminate asbestos fibres from any other types it is also found that an optical fibre count made on an environmental sample usually yields a definite value, but that this is totally unrelated to the presence or absence of any asbestos. The results of an optical fibre count performed on a general environment sample can be challenged in two ways: if the result is low it does not give confidence that asbestos is not present, since any asbestos fibres would have diameters too small for detection; if the result is high, the argument can be introduced that few, if any, of the fibres were actually asbestos and that they were all some other "fibrous" species. It might be argued that phase contrast optical fibre counts are the only ones for which reliable epidemiology data exists. However, since the fibres counted optically in an environmental sample are not usually asbestos, this argument is difficult to support. Accordingly, the phase contrast optical method is accepted as totally unsuitable for environmental sampling. NIOSH, whose optical membrane method is used for workplace monitoring in the U.S.A., recommends against the use of optical techniques for environmental studies.[16] Many others also advise against phase contrast microscopy for environmental measurements whether inside[17] or outside buildings, or for water samples.[18,19,20]

TABLE 2

EXFOLIATION PLANT AIR SAMPLING STUDY

Phase Contrast Optical Fibre Counts

Sample	Sampling Time, Minutes	Air Volume m^3	Fields of View Counted	Fibres* >5 µm Observed	Fibre* Concentration Fibres/mL
Static 2, Run 2	61	0.610	34	31	0.12
Static 1, Run 3	30	0.300	38	39	0.31

*Fibres are defined as all fragments having aspect ratios greater than 3:1, with lengths exceeding 5 µm and having diameters smaller than 3 µm.

Electron Microscope Fibre Counts

Sample	Sampling Time Minutes	Air Volume, m^3	Fibre† Concentration, Total Fibres/mL	Fibre† Concentration†† Fibres (>5 µm)/mL
Static 1, Run 2	60	0.60	0.024	<0.024
Static 2, Run 3	30	0.30	0.240	<0.047

†Fibres reported were those which an experienced operator may classify as chrysotile on the basis of morphology alone. It was not possible to confirm the identity of these by electron diffraction.

††No chrysotile fibres longer than 5 µm were detected, hence the airborne concentration can only be stated as less than that corresponding to one fibre. No amphibole fibres were detected, and concentrations of these were therefore similarly below the values specified.

3.2 Transmission Electron Microscopy (TEM) Methods

A modern transmission electron microscope (TEM) has a resolution of about 0.2 nm, which is more than adequate for resolving the smallest fibres of chrysotile which are about 40 nm in diameter. The image obtained also permits observations of some internal structure of each fibre. Chrysotile asbestos fibres have a tubular appearance on which identification can be based. This appearance is a result of the scrolled sheet structure of the individual fibre. However, a tubular appearance is not unique to chrysotile; halloysite and palygorskite can exhibit a similar appearance.[21] It has also been shown recently that vermiculite plates can roll into a scroll structure and also give rise to a fibre-like particle which can be confused with chrysotile.[22]

Identification of individual fibres can be achieved using selected area electron diffraction (SAED), in which a pattern characteristic of the crystalline structure of the fibre is produced. On a modern instrument, facilities are available for tilting and rotating the sample so that the crystalline fibre may be examined along its principal crystallographic directions. Switching between the image and the electron diffraction mode is accomplished simply by adjustment of lens currents and apertures. In investigations where identification of a specific mineral is required, computer analysis of electron diffraction patterns obtained along several principal directions of the crystal is necessary. However, since the crystal structures of tremolite, actinolite, crocidolite and amosite are practically identical, it is not possible using electron diffraction alone to discriminate between these minerals.

Discrimination between the amphibole asbestoses can only be reliably achieved by measurement of the chemical compositions of the fibres. Energy dispersive X-ray equipment (EDXA) which can be incorporated in the TEM allows this composition data to be obtained for each fibre in addition to the diffraction patterns. Although it is not practical or economical in most analyses to utilize both techniques on each suspected fibre, in many cases additional confidence in the identification is required. However, the amphibole character of the fibre must first be demonstrated by electron diffraction and this classification further

refined by the chemical information. The chemical information by itself is totally inadequate to permit classification of a fibre as amphibole. Indeed, the chemical information alone even for chrysotile asbestos is inadequate for classification, in that the particle may be a fragment of lizardite or antigorite which have similar compositions.

3.2.1 TEM Specimen Preparation Techniques

Early criticisms of TEM analyses for asbestos have now been shown to be unjustified and good intra and inter-laboratory agreement of results when using recognized techniques has now been established.[23] The agreement between laboratories was achieved without the introduction of direct personal consultation.

Much of the early difficulty was associated with specimen preparation techniques which incurred various degress of fibre loss. The carbon-coated Nuclepore technique now forms the basis of both the U.S. EPA* interim procedure for water samples[19] and the U.S. EPA procedure for air samples.[24] The Nuclepore filter is selected for this technique since its surface is relatively featureless apart from the cylindrical pores. The Nuclepore filter is somewhat unsuitable for field application. However, where the sample can be collected and hand-carried to the laboratory, direct sampling onto a Nuclepore filter remains the most suitable technique.

The steps in the technique are illustrated in Figure 3. After collection of the sample on a Nuclepore membrane filter, a carbon film is applied by vacuum evaporation. This envelops and traps all of the particles on the filter surface. A portion of the coated filter is then dissolved in chloroform, leaving a thin film of carbon which contains the particulate. The sample obtained is a copy, or replicate, of the filter surface with all of the original particles retained in position. The solvent extraction does not cause loss of particles, since absence of a particle would usually be indicated by a replicated region with no particle inside it.

* United States Environmental Protection Agency

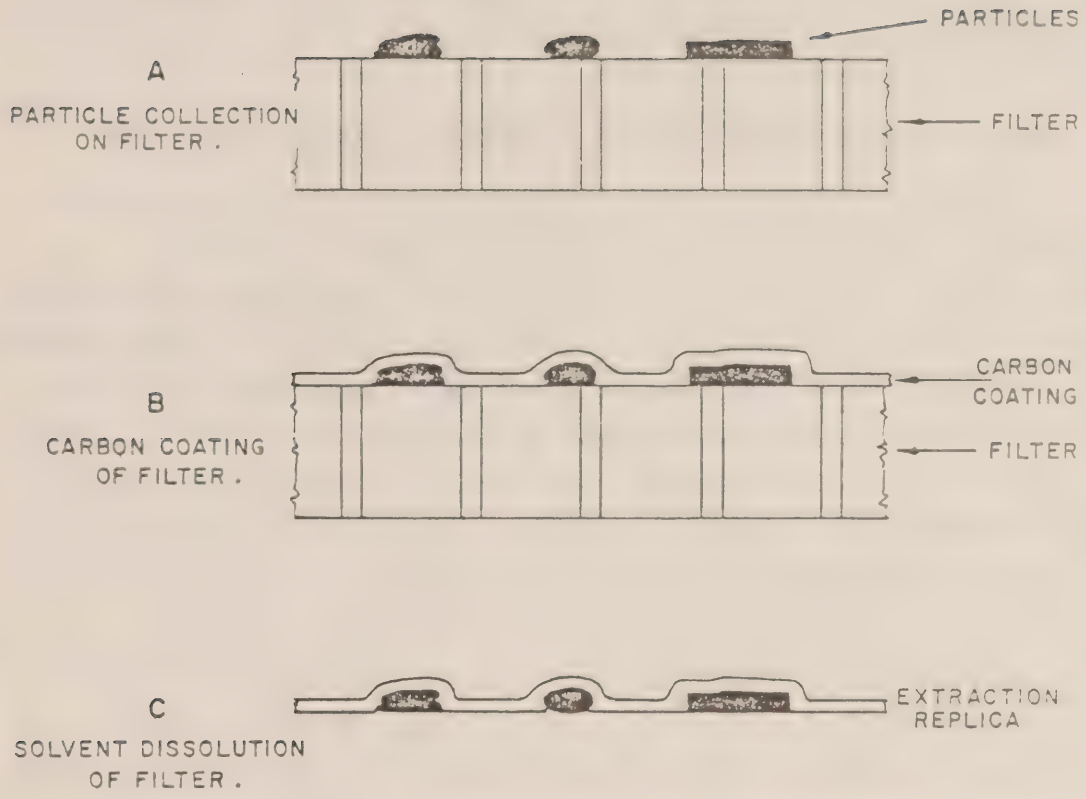


Figure 3. Steps in the carbon-coated Nuclepore procedure for TEM specimen preparation.

3.2.2 TEM Fibre Measurement

After preparation, fibres are then counted on the electron microscope sample in much the same way as they were in the optical technique, except that each fibre is identified and only asbestos is measured and incorporated in the count. The measurements can be made with detection levels of about 0.001 fibre/mL for *total* fibres regardless of their dimensions. The method of fibre identification is usually by electron diffraction; if necessary this is supplemented by EDXA measurements of the chemical composition.

3.2.3 Definition and Identification of Asbestos Fibres in the TEM

Positive identification of chrysotile by its morphology and SAED pattern is relatively simple if such a pattern is obtained. In Ontario, positive identification of chrysotile is accepted on the basis of tubular morphology alone, provided that some fibres of similar morphology in the same sample have been identified by their SAED patterns. A similar degree of confidence in identification of chrysotile can also be obtained on the basis of tubular morphology combined with the chemical composition. The identification of individual amphiboles, however, is much more involved. Apart from the elongated shape, there are no useful morphological characteristics, although there may be evidence of splintering. Once having located a suspected amphibole fibre, the SAED pattern is then inspected. If this is visibly recognizable as similar in spacing, orientation and character to that from known reference fibres, identification is accepted as positive in the interim procedure for water issued by the U.S. EPA.[19] However, in a sample about which nothing is known, the routine SAED pattern with a 5.3 Å spacing shows only that the fibre may be a chain or layer silicate. Some clay minerals, such as palygorskite, also give this spacing. Use of the routine SAED technique by itself is more a *rejection* technique than actual identification, since obviously non-amphibole patterns are frequently obtained and the fibre is rejected. Similarly, the EDXA spectrum showing a peak from silicon with various combinations of peaks from sodium, magnesium, iron and

calcium is not sufficiently specific for identification of amphiboles. A number of other minerals also have these approximate compositions. Positive identification of an amphibole fibre can only be achieved by a significant labour expenditure on each suspected fibre. The use of zone axis SAED patterns with computer analysis to determine their consistency with the amphibole crystal structure is the only way in which the presence of amphibole can be positively confirmed. Complete discrimination between the individual amphiboles requires matrix corrected EDXA measurements so that accurate compositional data are available in addition to the crystallography data.

A further complicating factor in the case of amphiboles is the use of the 3:1 aspect ratio as a definition of asbestos fibres. The 3:1 aspect ratio definition was adopted by various agencies for use in electron microscope counting techniques. Unfortunately, so many minerals cleave readily into such fragments that the application of environmental standards incorporating a 3:1 definition would result in closure of many mining and quarry operations. The U.S. Bureau of Mines has reported that the re-definition of the fibre to include only those particles having aspect ratios greater than about 10:1 would eliminate many ambiguities of identification between amphibole asbestoses and their non-fibrous polymorphs.[25] However the attitude of the Health Agencies is that such a re-definition is undesirable unless it can be demonstrated that those fibres having aspect ratios between 3:1 and 10:1 are harmless.

The amphibole asbestoses are not the only fibrous minerals in which there is a fibre definition problem. In the case of chrysotile asbestos, the adoption of a 10:1 aspect ratio definition would not significantly change the results obtained by electron microscopy, since most of the fibres encountered in the electron microscope exceed this value. There is, however, a major

problem because of variations of crystallinity which occur in this mineral often related to variations of the magnesium content. Fibres may have originated from known chrysotile, but subsequent chemical or thermal treatment can modify the composition or the crystallinity. A fibre may display the scroll structure of chrysotile, but no SAED pattern may be obtained. The fibre can then be shown to contain magnesium, silicon and iron. At what value of magnesium content do we classify the fibre as chrysotile asbestos? Scrolls of magnesium aluminum silicate have been encountered in association with vermiculite generated as a consequence of detachment and rolling up of a layer of the platy material. Do we regard this material as asbestos, and if so, how many turns on the scroll are necessary before we change our classification from platy to fibrous? Certainly, the scrolls are quite distinguishable from chrysotile by careful work on the TEM.

It is doubtful whether these definition problems will be solved quickly. More medical data are required to give guidance. The works of Stanton[5] and Pott[6] are already providing some indications that fibre dimensions may be the most significant parameters in relation to the health effects, and perhaps a meaningful fibre definition will emerge from these investigations.

4. ACCURACY OF FIBRE COUNTING TECHNIQUES

4.1 Fundamental Limitations

The counting of fibres on a sample is subject to statistical fluctuations, and these give rise to a fundamental limitation of the precision of the final result. In order to establish the precision of such measurements it is important to understand the statistics involved.

Random deposition of particles onto a filter during sampling is often stated to lead to a Poisson frequency distribution. However, particularly in the case of chrysotile, it has been found that the spread of the data may be two or

three times that which would be forecasted on the basis of the Poisson distribution. Since on any one sample there are usually inadequate data collected to demonstrate the actual distribution of the fibres, it has been found that the normal approximation is a more suitable one since the variability of the fibre density itself defines the spread of the data. The calculated confidence limits *are independent of the instruments in use*, and are primarily a consequence of the observed variability of the fibre density count at different locations on the sample filter. Specifically, if we examine and count fibres on 10 areas of the sample, what does this tell us about the range within which the actual particle concentration lies? We also have to ask the question whether the fibres are randomly and uniformly distributed on the filter.

If identification problems are discounted, the precision of the final answer depends primarily on the uniformity of the deposit and the number of fibres counted. As a rule of thumb, if about 100 - 200 particles are counted, at 95% confidence, a range in the measurement of ±30% is unavoidable. In practice, subjective decisions when using any microscope technique may lead to an additional spread in the results. It is most important that the user of reported results be aware of this significant range, and *confidence intervals should always be specified regardless of the fibre counting technique in use.*

4.2 Fibre Counting Criteria

There is currently no International Standard concerning the criteria for fibre counting using either optical or electron microscopy. This difficult topic is concerned with defining the way in which specific arrangements of fibres, overlapping or perhaps split fibres are counted. In any one group of individuals, different opinions as to how to count a specific arrangement will arise. Indeed, as stated previously, if the problem gets too difficult the most common rule is to move to another area and try that. However, such a practice obviously biasses the fibre count data towards the low side and this is somewhat undesirable. Therefore, there is a requirement to define standard fibre counting criteria for both optical and electron optical methods.

5. ASSESSMENT OF BUILDING INSULATION

5.1 Sampling Procedures

Building insulation is usually a very inhomogeneous material. In particular, in the common mineral wool-chrysotile asbestos mixtures, a volume of 1 cc may contain only mineral wool and the next 1 cc may contain a large proportion of chrysotile asbestos. Hence indications are that samples of building insulation must be much larger than this in order to be representative. Samples should always be collected through the total thickness of the sprayed layer, including material down to the underlying surface. The sampling procedure should only be performed by individuals who are experienced in the use of building insulation and who are able to recognize materials which may contain asbestos. The question then arises as to how many samples should be taken to determine whether or not asbestos is present in the sprayed insulation of a building. Table 3 shows the results obtained on samples collected from the Bowmanville High School, mentioned at the last public meeting of this Commission. In a total of 170 samples analyzed, 82 contained no asbestos at all and the remainder had concentrations ranging from 1% - 40%. By the collection of so many samples, it was possible to define regions of the building which required removal of insulation and other regions where no asbestos was present. However, the example is quoted to indicate the extreme inhomogeneity which existed within this building. Indeed, of the first three samples analyzed, only one contained asbestos. On the basis of a few samples it would have been possible to overlook the presence of asbestos within this building.

It is necessary at this point to develop a code of practice for sampling of bulk insulation in order to give reasonable confidence that any asbestos present has been discovered. The procedure developed by the U.S. EPA[26] specifies collection of one sample for each 5000 square feet of insulation spray. In most case this would lead to a large number of samples for analysis. However, if the decision has already been made to remove asbestos if present, analyses of this large number of samples will not change the action once asbestos has been detected in one sample. The Bowmanville example indicates that to base a decision on the results from one sample of insulation for a particular building is also an unacceptable procedure.

TABLE 3

RESULTS OF BULK INSULATION ANALYSIS

BOWMANVILLE HIGH SCHOOL

170 Samples Analyzed

Chrysotile Asbestos Concentration %	Number of Samples Analyzed
0	82
<1	5
1 - 5	6
5 - 10	4
10 - 20	33
20 - 50	40

It is important to understand that the mere presence of asbestos does not in itself provide sufficient information to determine if a possible asbestos hazard exists. It is necessary to consider the nature of the asbestos containing material and its use in the building. The USEPA considers the following factors important;friability, accessibility, exposed surface area, asbestos content, presence in air plenum or air stream, condition of material, and obvious water damage. These factors can readily be assessed by someone experienced in asbestos site inspections and the decision should be made on this recommendation.

5.2 Analytical Techniques

A number of analytical techniques have been suggested for analysis of insulation samples. These techniques include polarized light microscopy (PLM), dispersion staining optical microscopy, scanning electron microscopy (SEM), transmission electron microscopy (TEM), X-ray diffraction (XRD), and infrared absorption analysis (IR). Unfortunately, during the early requirements for this type of analysis, a number of inappropriate techniques have been used with disastrous results.[27] In one case, the material was dissolved and atomic absorption analysis performed for magnesium: chrysotile asbestos was confirmed. In fact, the material contained no asbestos and was composed solely of mineral wool (which contains magnesium) and gypsum. The infrared analysis technique is not suitable for analysis of insulation mixtures for asbestos because of the difficulties presented by other interfering species.[28] There is also a difficulty of interpretation when mixtures of asbestos varieties are present.

The inhomogeneity of insulation has important consequences for the utility of precise quantitative measurements. There is little purpose to be served by a measurement of the asbestos concentration in one sample to a 1% accuracy if the material contains 40% asbestos at one location and 15% or 10% at the next. In such a situation, better information on which to base a decision is provided by analysis of a greater number of samples by a simple technique than by a single sample analysis of high precision and correspondingly high cost.

The U.S. EPA[26] has specified the use of polarized light optical microscopy; where a supportive technique is required, X-ray diffraction is suggested. The PLM technique requires considerable expertise. However, it should be recognized that X-ray diffraction used as a routine screening method may fail to detect as much as 5% asbestos,[3] and in the case of chrysotile, may fail to detect even higher concentrations,[29] particularly if the chrysotile is originally lacking in crystallinity or has been milled.[30]

At ORF we have followed the lead of the EPA in the use of polarized light optical microscopy but we have supplemented the identification techniques with dispersion staining optical microscopy and SEM-EDXA analysis. The various fibrous components of insulation materials such as mineral wool and cellulose can readily be discriminated from asbestos fibres by PLM in the hands of a competent microscopist. However, the non-fibrous binding materials often attached to the fibres make identification more difficult, and it is then necessary to resort to the SEM for confirmation of the identification. In severe cases it may be necessary to use chemical treatment to remove the interfering materials, but such treatments can change the optical properties of the fibres. Quantification of the proportion of asbestos is achieved by visual estimation, although an interim method recently published by Research Triangle Institute[31] recommends a point counting technique to assist in more objective determinations of asbestos content where this is considered necessary.

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APPENDIX B

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A REVIEW OF DIAGNOSTIC TESTS FOR CAUSATION

Prepared for the Royal Commission on Asbestos
Second Public Meeting, December 12, 1980.

Introduction:

Two different species of expertise are required for understanding the impact of asbestos on the health and safety of Ontarians. The first is biologic expertise in the subject matter of asbestos and its effects on cells, tissues, organs and organisms. The second is methodologic expertise in the critical assessment of data concerning etiology or causation.

As one whose research and clinical work is focused in cardiovascular disease, I have no expertise in the biology of asbestos-related disorders. Rather, I have been asked to introduce certain principles pertinent to the critical assessment of data concerning causation, especially as it relates to dose-response, thresholds, and safe thresholds. I shall do this within the broader context of a set of "diagnostic tests for causation." The examples or "cases" I shall use are deliberately chosen from several fields, for I wish to emphasize the "transferability" of these ways of looking at evidence. For ease in tracking them further, I have taken most of them from The Canadian Medical Association Journal.

Case-Presentations:

Perusal of a grab sample of back issues from Volume 121 of The Journal reveals that its clinical readers are faced with claims about the etiology and causation of human illness every time they read. For example, on page 291 they are warned about dietary fibre and colon cancer (4); on page 404 cimetidine is implicated as a cause for diminished libido (5); on page 548 an effect of cigarette smoking on the risk of occupational lung cancer is proposed (6); on page 706 heavy tea consumption is blamed for iron deficiency (7); 10 pages later readers learn about drugs that cause dependence (8); on page 1060 viral encephalitis is proposed as a cause for Huntington's chorea (9); on page 1361 they are told that malfunctioning brown fat may be a cause for obesity (10); 16 pages later Campylobacter is implicated in the etiology of ileocolitis (11); on page 1566 they are asked to reconsider whether colour-blindness in auto drivers causes traffic accidents (12); finally, on page 1580 the roles of cold snaps and snow shovelling as causes for sudden death are debated (13).

These 10 "cases in causation" have important implications for both private and public practitioners, because they are accompanied by some powerful recommendations: We should try to change our patients' eating habits, have second thoughts about prescribing some specific drugs, discourage tea drinking in certain native groups, consider (or reconsider) banning colour-blind drivers, and have lots of patients quit shovelling snow.

When we add to the foregoing the bevy of claims for causation that the general public, to their distress, come upon in the lay press and on television, it becomes clear that all of us are forced to make judgements and to give advice about causation all the time.

To help meet these demands for instant sagacity, we have brought together some "applied principles of common sense" that should help both private and public health practitioners assess an article that claims to show causation. They are distilled from the work of a number of methodologists, most notably Austin Bradford Hill (14).

The application of these common sense principles involves two steps. First, readers should scan the Methods section of the article to see whether the basic methods used were strong or weak. Second, readers should then apply a set of "diagnostic tests" for causation to the remainder of the article.

STEP ONE: DECIDING WHETHER THE BASIC METHODS USED WERE STRONG OR WEAK

Sometimes you can identify the basic method used in a study from its title; other times you must examine its abstract or Methods. Thus, step one can be accomplished quickly, without having to read the Introduction or Discussion. This step is summarized in Table I.

Suppose we really wanted to find out whether snow-shovelling was a cause for heart attack in middle-aged (your age plus 5 years) men. What would be the most powerful sort of study we could find in the clinical literature?

Most of you, we hope, would start by looking for a true experiment in humans: a study in which middle-aged men would have been randomly allocated (by a system analagous to tossing a coin) to habitually shovel or not shovel snow each winter*, and then followed to see how many in each group went on to sudden death. Evidence from such a randomized trial is the soundest evidence we can ever obtain about causation (whether it concerns etiology, therapeutics or any other causal issue), and the reasons for this, if not already clear, will become apparent as we proceed. The basic architecture of the randomized trial is shown in Table II.

Although the true experiment (randomized trial) would give us the most accurate (or valid) answer to a question of causation, and therefore represents the strongest method, we will not find it very often in our clinical reading. In many cases (including the present example) it is not feasible to do a randomized trial to determine etiology, and in some it is downright unethical; for example, who would ever consider carrying out a true experiment that would deliberately cause viral encephalitis in a random half of a group of humans to see whether they were rendered more likely to develop Huntington's chorea (9)?

Thus, we are much more likely to encounter sub-experimental studies into the risk of heart attack from snow-shovelling. For example, the next most powerful study method (the cohort study)would identify two groups (or cohorts) of middle-aged men, one cohort who did and the other who did not make a practice of shovelling snow each winter. Such a study would then follow these two cohorts forward in time, counting the heart attacks that occurred in each. In this case, the direction of inquiry is forward in time, as depicted in Table III. If the heart attack rate was

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* Those who balk at the feasibility of this approach should recognize that the point at issue here is validity, not feasibility. On the other hand, the authors could have provided the controls with snow blowers!

higher in the cohort who shovelled snow, this would constitute reasonably strong evidence that snow-shovelling precipitated heart attacks. However, the strength of such a cohort analytic study is not as great as that of a randomized trial, and the reason for this difference in strength is apparent if we consider the middle-aged man with angina pectoris. First, is he more likely to avoid snow-shovelling or other activities that precipitate angina than his angina-free neighbour? Yes. Second, is he at higher risk than his neighbour for heart attack? Yes again. Thus, the cohort analytic study could provide a distorted answer to the causal question if men at high risk of heart attack for extraneous reasons* were not equally distributed between the cohorts of those who did and did not shovel snow. We see, then, that we must view a sub-experimental study such as the cohort analytic study with some caution and suspicion.

A second type of sub-experimental study deserves even greater caution in interpretation, and this is the case-control study. In a case-control study, the investigator gathers "cases" of men who have suffered a heart attack and a "control" series of men who have not had a heart attack. Both groups of men are then questioned about whether they regularly shovel snow each winter; if those who had heart attacks were more likely to be regular snow shovellers, this would constitute some evidence, though not very strong, that snow shovelling might cause, or at least precipitate, heart attack. Thus, in this case the direction of inquiry is backwards in time, as shown in Table IV.

Why is the case-control analytic study low on the scale of strength? This is because it is so very liable to bias. Not only is the case-control study susceptible to bias from the angina patient we noted in the cohort study; the case-control study is susceptible to several other sorts of bias (15). For example, if snow-shovelling precipitated not only heart attack but sudden death, many victims would not survive long enough even to be included in a case-control study, much less interviewed. As a result, snow-shovelling would appear to be a benign pasttime when, in fact, it was lethal for some middle-aged men. For this and other similar reasons, the results of case-control studies are tenuous at best in sorting out the etiology and causation of human illness.

One final type of sub-experimental study deserves mention. This is the case-series, in which an investigator might simply report that 60% of his male heart attack patients were shovelling snow just before onset of their infarcts. No comparison group is provided, and about all that the reader can conclude is that heart attack can (but not necessarily does) follow snow shovelling. Such case series, though often thought-provoking, are prone to over-interpretation, especially by their authors. In terms of strength, case-series are best used to stimulate other, more powerful investigations. All too often, however, they provoke authoritarian (rather than authoritative) clinical advice about etiology, prevention and therapy.

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* You may come upon the term confounder in your reading, and that's what angina is in this example. First, it is extraneous to the question posed (show-shovelling); second, it is a determinant of outcome (heart attack); finally, it is unequally distributed between the cohorts of exposed and non-exposed persons.

In summary, then, readers of reports purporting to show etiology or causation should begin by deciding whether the basic methods used were strong or weak (Table I). If the basic method was a randomized trial, it is the strongest and usually can be trusted. This is how, for example, the best evidence was obtained on the real side-effects produced by frequently used antihypertensive drugs (16). A cohort analytic study, although weaker than a randomized trial, is always preferred over a case-control study, and can sometimes be trusted. Thus, the most convincing (but nonetheless disputed!) evidence about the possible side-effects of oral contraceptives comes from a large cohort study carried out by British general practitioners (17). The case-control study is a weak design, and has often led to erroneous conclusions (such as the now discredited link between reserpine and breast cancer (18)); however, for some extremely rare disorders (especially rare adverse drug reactions) we may only have case-control studies to go by and may be forced, however reluctantly, to trust them. Finally, it is not possible to tell whether any given case series can, all by itself, be trusted on an issue of etiology or causation. Thus, if other, stronger evidence is available, such case-series should be passed over.

STEP TWO: APPLYING THE DIAGNOSTIC TESTS FOR CAUSATION

Having decided from the foregoing that the article warrants further consideration, the reader should then turn to the Results the Introduction and the Discussion to see how the data fit some commonsense rules of evidence. In making this causal decision, information should be sought relative to the diagnostic tests that are listed in Table V. They are discussed in order of decreasing importance and we have suggested their impact upon the causal decision in Table VI.

The rules for interpreting clinical diagnostic tests we described in an earlier Rounds in this series (2) can be applied here as well. For example, some of these tests for causation (such as evidence from randomized trials) are more accurate than others (such as analogy). Furthermore, many of them (such as temporality) are better for "ruling out" causation than for "ruling it in." Finally, biologic sense and epidemiologic sense, although prominent in many articles, are low on the list. This is because they have relatively low specificity; it is possible to "explain" almost any set of observations.

1. Is there evidence from true experiments in humans?

As we explained earlier, these are investigations in which identical groups of individuals, generated through random allocation, are and are not exposed to the putative causal factor and are followed for the occurrence of the outcome of interest.

As we have just seen, this is the best evidence we will ever have, but it is not always available and is rarely the initial evidence for causation. Nonetheless, any consideration of an issue in causation should begin with a search for a randomized trial.

2. Is the association strong?

Strength here means the odds favouring the outcome of interest with,

as opposed to without, exposure to the putative cause; the higher the odds, the greater the strength.

There are different strategies for estimating the strength of association. In the randomized trial and cohort study (Tables II and III) patients who were, and were not, exposed to the putative cause are carefully followed up to find out whether they develop the adverse reaction or outcome. Such a cohort study would, for example, compare the occurrence of impotence among ulcer patients who did, and did not, receive cimetidine (5).

Cohort studies (Table III) are methodologically attractive because, like the randomized trial, they permit direct calculations of strength (relative risk) by comparing outcome rates in exposed and non-exposed persons:

$$\frac{a}{a+b} \quad \cdot \quad \frac{c}{c+d}$$

However, as we learned in the previous section, cohort studies are often lengthy and expensive. Accordingly, the greater speed and lower cost of the case-control study (Table IV), in which patients with and without the outcome of interest (e.g., impotence) are selected and tracked backward to their prior exposure to the putative cause (e.g., cimetidine), make it a much more popular approach, particularly as the first step in probing the conclusions of initial case-series. Case-control or "trohoc" (19) studies pay a methodologic price for their savings in time and dollars. Strength or relative risk can only be indirectly estimated:

$$\frac{ad}{bc}$$

a calculation which, though justified algebraically, is viewed with some skepticism (19).

Moreover, as we have seen, case-control studies are particularly vulnerable to a series of systematic distortions (biases) which may lead to erroneous estimates of the strength of association and, therefore, incorrect conclusions about causation. Some of these biases were discussed in the previous Round in this series (2) and still others are described in detail elsewhere for readers who want to pursue this further (15).

A review of the potential effects of these biases in distorting the conclusions of case-control and cohort studies leads to two conclusions: First, the case-control studies are subject to more sources of bias than are cohort studies. Second, whereas one can usually anticipate and overcome (through appropriate methods, rigorously applied) those biases affecting cohort studies, this solution is either much more difficult or impossible in the case-control stretegy. As a result, readers can place considerable confidence in estimates of strength from s randomized trial, fair confidence in an estimate of strength from a cohort study and only a little confidence in an estimate of strength when it comes from a case-control study.

3. Is the association consistent from study to study?

The repetitive demonstration by different investigators of an association between exposure to the putative cause and the outcome of

interest, using different strategies and in different settings, constitutes consistency. Thus, much of the credibility of the causal link between smoking and lung cancer arises from the repeated demonstration of a strong statistical association in case-control, cohort and other study designs.

4. Is the temporal relationship correct?

A consistent sequence of events of exposure to the putative cause, followed by the occurrence of the outcome of interest, is required for a positive test of temporality. Although this diagnostic test looks easy to apply, it is not. What if a second predisposing factor or a very early stage of the disorder itself is responsible both for exposure to the putative causal factor and for progression to the full-blown outcome? Indeed, such an explanation might apply to studies that have linked the use of illicit stimulant or depressant drugs to the subsequent diagnosis of psychosis or depression, respectively (20). Did the different illicit drugs cause specific forms of subsequently diagnosed mental illnesses, or did individuals with different sub-clinical but progressive mental illness seek out the specific drugs? This yardstick is understandably easier to apply to cohort than to case-control studies, since the latter can infer the temporal association between "exposure" and "outcome" only after both have occurred.

5. Is there a dose-response relationship?

The demonstration of increasing risk or severity of the outcome of interest in association with an increased "dose" or duration of exposure to the putative cause satisfies this diagnostic test. For example, in a report linking conjugated estrogens with endometrial carcinoma (21), the relative risk of developing endometrial cancer rose from 5.6 among those who used the drug 1 - 4.9 years to 7.2 among users for 5 - 6.9 years and, finally, to 13.9 among users for 7 or more years.

Reverse gradients are useful, too. Indeed, some of the most compelling evidence on the link between cigarette smoking and lung cancer is the progressive decline in cancer risk that has been reported as previous smokers celebrate successive anniversaries of their last cigarette.

Thus, the observation of a dose-response gradient provides support for the causal argument. Because this is a central issue for the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario, this text will consider four special questions concerning dose-response relationships:

(a) Does the presence of a dose-response relationship prove causation?

Although a dose-response relationship supports causation, it does not prove it (save in the unusual case of a true experiment in which different doses are randomly assigned to different subjects by the experimenter - a situation which is not acceptable when the exposure is judged to be potentially harmful). This is because the different doses

of the putative causal factor may simply be "markers" for another factor that is the true cause. For example, a dose-reponse relationship exists between matches and lung cancer, yet it is the cigarettes lighted by these matches that are the true cause. Similarly, dose-response relationships between the duration of exposure to some putative cause and an outcome of interest are confounced with age; those exposed longer are often also older. Thus, one must examine and critically assess does-response relationships to decide whether they represent causal factors or mere "markers." A family of statistical procedures are available that assist this assessment by adjusting for known confounders such as age, and will help reviewers draw conclusions that will not vary about phenomena that do vary.

(b) Does the absence of a dose-response relationship disprove cause?

Once again, the answer is no; the absence of proof is not the proof of absence. To be sure, the absence of a dose-response relationship supports a "not guilty" verdict; however, there are a number of reasons why a true dose-response relationship may be missed:

(i) The measurement of the dose of exposure may be beyond the capacity or control of the investigator, especially in backward-looking case-control studies. As a result, persons will be misclassified with respect to their actual dose received, and the results of this misclassification will be to obscure or even ablate the dose-response relationship. Thus, the problem lies in the quality of the data that were available or attainable, not in the appropriateness of the search for a dose-response relationship.

(ii) only certain sub-groups of exposed persons may be susceptible, due to their genetic make-up or to the presence of co-causal agents. If these sub-groups constitute a minority of exposed persons, their dose-response relationship may be swamped by the unaffected majority.

(iii) the outcome of interest may have several causes, of which the putative causal factor is only one. Again, in this circumstance a real dose-response relation may be obscured.

(iv) the range of doses considered may be too narrow to exhibit a gradient of the outcome of interest. Once again, the fault lies in the quality of data rather than in the decision to seek evidence for a dose-response relationship.

(v) finally, the dose or exposure measurement may capture only a single source of the putative causal factor and the study subjects may actually have received greater doses from other sources. Once again, a real dose-response relationship is obscured by data of relatively low quality.

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(v) finally, the dose or exposure measurement may capture only a single source of the putative causal factor and the study subjects may actually have received greater doses from other sources. Once again, a real dose-response relationship is obscured by data of relatively low quality.

(c) Do "thresholds" exist, below which variations in dose make no difference?

Discussions of this topic usually generate more heat than light. Evidence of thresholds below which variations in dose have no relation to effects have been suggested for several biologic and environmental factors; arguably, none have been convincingly demonstrated. This is because we cannot be sure that we have looked hard enough or long enough for the possibly small and quite delayed effects of low doses. A portion of the threshold debate concerns the shape and location of the dose-reponse relationship when it is charted on a grid.

The mathematical properties of such shapes and locations are complex, but a few simple rules will assist their interpretation by anyone:

(i) beware extrapolations beyond actual observations, for they represent acts of faith rather than acts of observation.

(ii) dose-response relationships that are "fit by eye" tell you more about their creators' understanding of science than about the nature of the problem under scrutiny.

(iii) a key element of such a chart is the location and contour of the dose-reponse relation at the point of zero dose. If the response at this point is zero or positive and the slope of the dose-reponse line or curve is positive, this suggests that there is no dose at which some response will not occur (no threshold exists). If on the other hand, the response at this point is negative or the slope of the dose-response line or curve is zero or negative, this suggests that there are doses at which no change in response is expected (a threshold does exist).

(d) Do "safe thresholds" exists?

On the basis of the answer to the previous question, the answer here has to be: in the absence of actual observations, carefully assessed, we simply don't know. But is this really the question we should be asking? Perhaps a more appropriate question is: Do "acceptable thresholds" exist? There is abundant everyday evidence that "acceptable thresholds" exist for all sorts of environmental hazards: highway speed limits are a case in point. Thus, the issue becomes one of reflecting society's judgements of the utility of the social risks (e.g., health) and social benefits (e.g., economic) of various points along the dose-response relationship.

Perhaps a useful analogy is found in medical practice, where we daily weigh the risks and benefits (both of which are often dose-related) of diagnostic and therapeutic manoeuvres for our patients. What we clinicians demand are objective data, carefully gathered, on the dose-response relationships for these risks. This requirement for objective, precise and accurate measurement of doses and responses, frequently over long time periods, is a major challenge both in the evaluation of clinical manoeuvres and in the study of environmental hazards. Data of a level of quality sufficient to answer the question posed was a prerequisite for bringing clinical medicine out of the dark ages. I suggest that the same applies to environmental medicine.

6. Does the association make epidemiologic sense?

This guide is met when the article's results are in agreement with our current understanding of the distributions of causes and outcomes in man.

For example, Freeman, when reviewing the possible role of dietary fibre in the pathogenesis of human colon cancer, noted several studies in which the distribution of dietary fibre among different geographic areas or population groups was inversely related to the occurrence of colon cancer in these same areas and populations (4). Recognizing the tenuous nature of such epidemiologic correlations (after all, the declining birth rate in Europe has closely paralleled the disappearance of storks from their cities), Freeman called for "longterm prospective studies" to better define the role of dietary fibres in human cancer.

7. Does the association make biologic sense?

Is there agreement with current understanding of the response of cells, tissues, organs and organisms to stimuli? It is with this yardstick that non-human experimental data should be measured. Although virtually any set of observations can be made biologically plausible (given the ingenuity of human mind and the vastness of the supply of contradictory biologic facts), some biologic observations can be compelling, such as Himms-Hagen's description of the production of massive obesity in certain strains of mice whose brown fat has only a limited capacity for thermogenesis (10).

8. Is the association specific?

The limitation of the association to a single putative cause and a single effect satisfies this diagnostic test. Examples here are some of the highly characteristic genetic disorders in which derangements in a single enzyme or other protein produce quite specific illnesses such as hemophilia A or cystinuria. One of the minor diagnostic tests, this is only moderately useful, and then only when it is present. The weakness of this test is underscored when considering teratogens, where multiple effects in several organ systems are commonplace.

9. Is the association analagous to a previously proven causal association?

The last and least of the diagnostic tests, this yardstick would link the scrotal cancer of chimney sweeps in a former era with the more recent appearance of lung cancer among those who inhale, rather than wear, the products of combustion.

USE OF THESE GUIDES TO READING

When confronted by a question of causation, these nine diagnostic tests can be used to distill one's clinical reading and, with the assistance of judgements such as those shown in Table VI, reach a causal conclusion. Even before reading, these guides can be used to increase the efficiency of a literature search, focusing attention on those publications which will shed the strongest light on the causal question and warning the user against accepting plausible but biased conclusions.

Even after extensive reading and the application of all nine diagnostic tests, however, you may remain uncertain about whether, for example, exposure A really causes illness B. What do you do then, and how do you translate all of this deliberation into clinical action?

We suggest, as shown in Figure 2, that this "decision for action" has two components. First is our certainty about causation, based upon the application of the nine diagnostic tests for causation to our consideration of the consequences of the alternative courses of action that are open to us (recognizing that these courses of action include non-interference as well as maintaining the status quo). The decision for action results from the interplay of these two components. Consider two examples:

The three reports indicting reserpine as a cause of breast cancer that appeared abruptly in 1974 (22-24) precipitated a crisis in the management of hypertension. How were we to advise and treat patients whose high blood pressure was kept under control with this drug? The first component of this decision considered the degree of certainty that reserpine did, indeed, cause breast cancer; it was never very great [and in fact the drug was later virtually pardoned by some of its earlier accusers (25)]. On the other hand, the second component of this decision identified an alternative course of action that was highly attractive to many Canadian clinicians: switching appropriate patients from reserpine to propranolol. Thus, in this case even a low degree of certainty about causation was attended by a clinical decision to take many patients off a drug because an alternative treatment was available.

By contrast, the degree of certainty that oral contraceptives cause thromboembolism is much higher. Nonetheless, oral contraceptives remain in widespread use. Although the reasons behind this decision to continue oral contraceptive use in the face of growing evidence that it causes thromboembolism are complex, it is due in part to the second component of the decision: the consequences of alternative approaches to birth control may be judged even less desirable than the small but real risk of thromboembolism. Thus, the use of oral contraceptives continues (and, interestingly, the diagnostic test of the dose-response gradient is involved to justify the progressive reduction of certain hormonal constituents of the oral contraceptive pill).

The diagnosis of causation is not simply arithmetic, and the strategies and tactics for making this judgement are still primitive. The diagnostic tests presented here are a start, and it is suggested that their use, particularly when clearly specified before a review of relevant data, will lead to more rational - albeit less colourful - discussions of causation in medicine.

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Figure 2: A Clinical Decision for Action

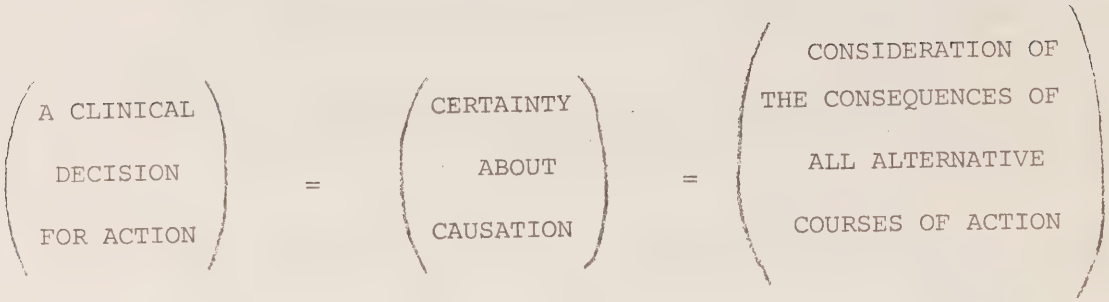

TABLE I: Step One: Deciding Whether the Basic Methods Used Were Strong or Weak

STRENGTH	METHOD
Strongest	Randomized Clinical Trial
	Cohort Study
	Case-Control Study
Weakest	Case-Series

TABLE II: A Randomized Trial

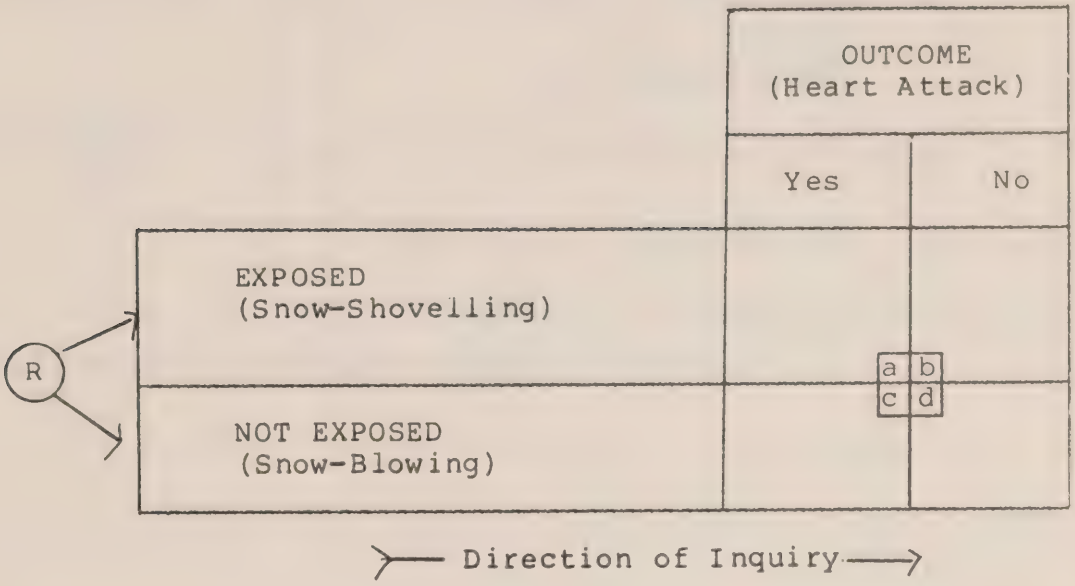

TABLE III: A Cohort Study

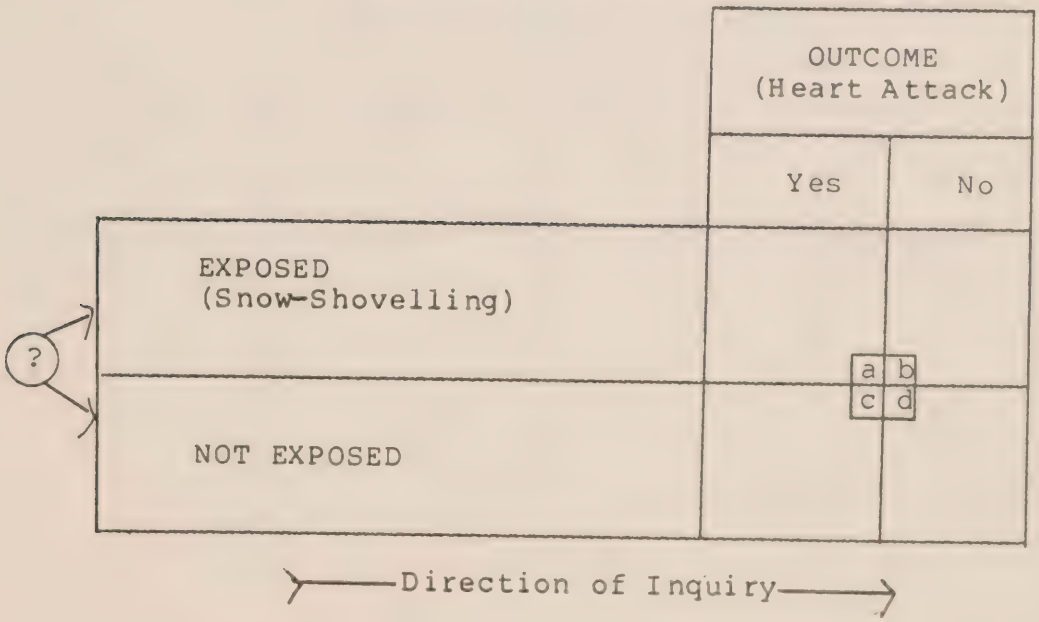

TABLE IV: A Case-Control Study

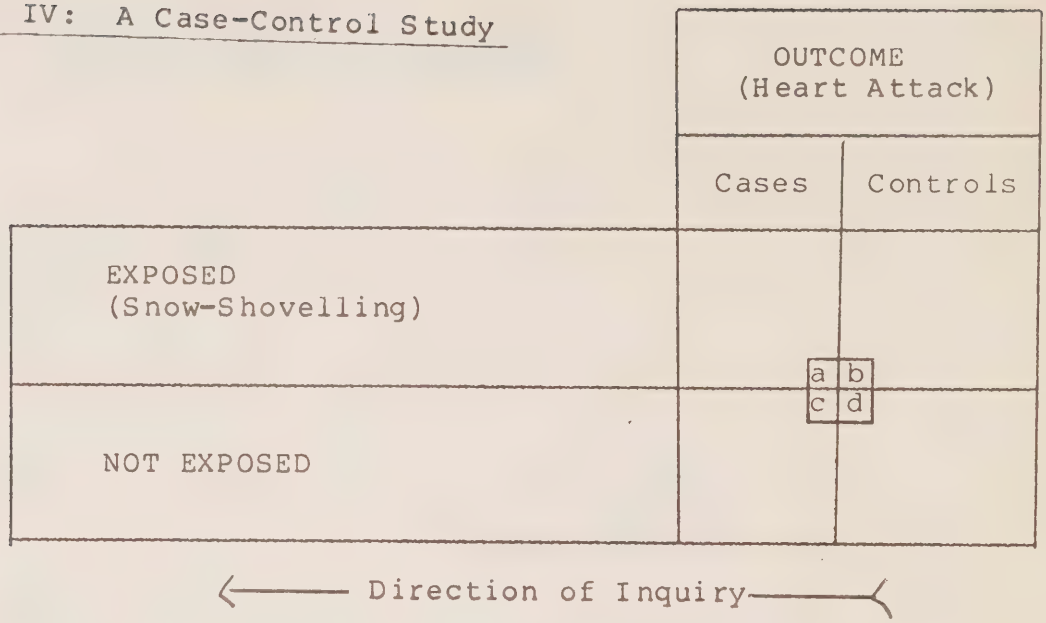

TABLE V: Step Two: Applying the Diagnostic Tests for Causation: *

1. Is there evidence from true experiments in humans?
2. Is the association strong?
3. Is the association consistent from study to study?
4. Is the temporal relationship correct?
5. Is there a dose-response gradient?
6. Does the association make epidemiologic sense?
7. Does the association make biologic sense?
8. Is the association specific?
9. Is the association analagous to a previously proven causal association?

* These diagnostic tests are listed in decreasing order of their importance.

TABLE VI: Importance of Individual Diagostic Tests in Making the Causal Decision

	EFFECT OF TEST RESULT ON CAUSAL DECISION**		
DIAGNOSTIC TEST*	TEST RESULT CONSISTENT WITH CAUSATION	TEST RESULT NEUTRAL OR INCONCLUSIVE	TEST RESULT OPPOSES CAUSATION
Human Experiments	+ + + +	- - -	- - - -
Strength of Association			
- from randomized trials	+ + + +	- - -	- - - -
- from cohort study	+ + +	- -	- - -
- from case-control study	+	Ø	-
Consistency	+ + +	- -	- - -
Temporality	+ +	- -	- - - -

Gradient	+ +	-	- -
Epidemiologic Sense	+ +	-	- -
Biologic Sense	+	Ø	-
Specificity	+	Ø	-
Analogy	+	Ø	Ø

** Meaning of symbols:

+ = causation supported

- = causation rejected

Ø = causal decision not affected

The number of +'s and -'s indicates the relative contribution of the diagnostic test to the causal decision.

* These diagnostic tests are listed in decreasing order of their importance.

APPENDIX C

ADDRESS BY:

Dr. David Muir
Director
Occupational Health Program
McMaster University

THE HEALTH HAZARDS OF ASBESTOS: ASSESSING THE QUALITY OF SCIENTIFIC EVIDENCE

Prepared for the Royal Commission on Asbestos,
Second Public Meeting, December 12, 1980

The Royal Commission on Asbestos has asked me to consider the quality of evidence in relation to four specific questions. These are as follows:

(a) What is the shape of the dose/response curve for the health effects of asbestos exposure?

(b) Is there a threshold?

(c) Is there a safe threshold?

(d) How big is the health improvement from policy-induced reductions in exposure in the workplace or in public buildings?

I believe it will be helpful to confirm, in the first instance, that there is indeed a relationship between the intensity and duration of exposure and the health risk. The evidence for this is overwhelming and almost self-evident. The reason for emphasizing this point is that it has clear implications for the definition of health public policy. It is difficult to resist the proposal that any unnecessary exposure should be avoided. If this Commission identifies some airborne level at the workplace to be used for control purposes then work practices designed to reduce exposure still further would appear to be eminently desirable and should be identified clearly as such. This idea of emphasizing "good housekeeping" is likely to be of increasing value when we come to consider exposure in the public domain. Airborne fibre levels in such situations are generally an order of magnitude or more below those encountered in industries manufacturing asbestos products. I am unable to find evidence relating exposure to health at these low levels of airborne asbestos and doubt whether measurements of the airborne fibre concentrations contribute information are of practical value. The measurements themselves are exceedingly difficult to undertake and more difficult still to interpret.

The specific questions posed by the Commission form the central core of information required to frame policy. In attempting to obtain answers which will find wide acceptance in the community some fundamental difficulties must be resolved.

By far the most important of these difficulties is the fact that disease currently attributed to asbestos had its origins in exposure many years ago. This is particularly so with respect to mesothelioma. We have little or no evidence on the environmental conditions existing twenty to forty years ago although a number of estimates have been made and have been used in epidemiological analyses. While there can be little doubt that dust conditions were considerably higher before the introduction of control measures it may legitimately be asked whether these estimates are robust enough to serve for complex mathematical model building of the type popular with some epidemiologists.

The second major problem is to separate risks associated with different types of fibre. No doubt the Commission will be considering this aspect in detail and will have to decide whether there is some specific risk caused by amphibole fibre or whether the apparent special problems with these minerals stem from more intense airborne fibre levels associated with their use in

previous years. If specific effects due to the type of fibre are accepted then it becomes of considerable importance to identify the type of asbestos used in individual industries in the past. This is surprisingly difficult to achieve. A number of epidemiological claims have had to be modified after initial analysis when later evidence suggested that varying amounts of mixed fibres had been used but which did not appear on the main company records. This type of information may sometimes be more accurately obtained from the shop floor then from the accounting office.

The problem is even more complex then it appears. The evidence against crocidolite is sufficiently consistent that, if the Commission identifies a level for chrysotile to be used for monitoring in the workplace, then it is probable that there will be a good deal of support for the proposal that some yet lower level be required for amphiboles. In addition to this, however, there is an obvious problem in comparing the health experience of chrysotile miners in Quebec with insulation workers in New York. Assuming that estimates of previous exposure in the epidemiological studies concerned are relatively accurate then there appears to be very strong evidence that the health risk, as measured by generally used methods, depends on the specific industrial situation. This is almost certainly what common sense would predict. The size distribution of the fibres in the total airborne state varies accordingly to the industrial process and is well-known to so vary. It would be as unreasonable to assume that the health effect is unrelated to this variation as it is to assume that all asbestos species are biologically identical. The practical and economic importance of this is considerable to Canada. The apparent lack of health effects claimed for chrysotile workers in Quebec at relatively high levels of exposure is remarkable. We need to know why there was such a large contrast in their mortality experience compared to insulation workers.

Any epidemiological evidence presented in this difficult field requires critical analysis according to the concepts eloquently formulated by Dr. Sackett. Surveys conducted on selected groups of workers are more than useless and indeed are positively harmful as a result of the misleading information which they generate. Since asbestos-related diseases do not generally occur until many years after exposure begins and may appear many years after exposure ceases, then appropriate analytical strategies must be used. This implies following all exposed workers and studying their health experience in relation to exposure levels and time since onset of exposure.

Some of the asbestos literature does not accord with these concepts. The Commission could perform a valuable service by analyzing the available information according to its acceptability in modern epidemiological terms.

A major contribution could be made also by placing confidence limits on estimated exposure indices. When measurements are available these are often very approximate but are likely to be better than nothing. When no measurements at all are available, it is not unusual to discuss the problem with long-term employees and to obtain some opinion as to what it was like in "the old days." These early estimates have a considerable influence on the analytical results and any method of assessing their reliability would be invaluable. There is an enormous variation in the estimates of previous exposures. This leads to one argument to the effect that present disease

levels were caused by very high levels of dust exposure many years ago; that current levels are trivial by comparison and may therefore be viewed as relatively harmless. Another view claims that previous dust levels were not as high as all that and that dust levels currently prevalent are unacceptable. Hopefully, the Commission will undertake this review mindful of the possibility, indeed the probability, that any estimates of fibre exposure and of health effects are likely to be species-specific and to be industry-specific.

These analytical contraints suggest that replies to the questions posed by the Commission must be presented in a very conditional form since they are based on very limited published information relating mainly to chrysotile.

As far as asbestos and lung cancer are concerned, the evidence suggests that a linear dose-response relationship fits the data as well as anything. The choice of such a relationship having a zero intercept is to some extent a matter of mathematical convenience and may not necessarily describe the true biological response. It is difficult to extrapolate with confidence from the health effects observed at fairly high dust levels in order to predict the health outcome in workers exposed to dust levels outside the observed range. The fact that the intercept is at zero does not constitute evidence that very low levels necessarily represent a major health problem. In the same way, the fact that a given set of data may fit a line with a positive intercept is hardly good evidence that a so-called safe level can be identified. The value of fitting a dose-response curve lies in confirming that reducing dust levels is likely to have health benefits, and that low level exposure is likely to result in low level mortality.

The only really acceptable evidence of a no response or completely safe level must be based on observations of groups of workers who have been exposed to measured levels and who have been followed for sufficient time. In the chrysotile mining industry, it appears that remarkably high fibre exposure levels do not result in a detectably increased mortality. Much lower chrysotile levels certainly do appear to cause measureable mortality and morbidity in the fabricating and insulation industries, and the Commission will no doubt take these remarkable findings into careful consideration.

There is little evidence available to describe the dose-response relationships between asbestos exposure and the production of mesothelioma. It seems certain that there is an overall effect such that heavy exposure is more likely to cause mesothelioma and that this effect is species-specific. No zero effect has been identified but, again, this does not exclude the possibility that such a threshold may exist.

The real difficulty throughout the whole analysis is a lack of reliable data concerning the health effects of low level exposure. The confidence limits of the data are so wide that firm predictions are unlikely to be well-founded no matter who makes them. Some uncertainty would appear to be the most appropriate position. This uncertainty should not inhibit the decision-making process, but it is a good start to separate clearly between those things we really do know and those things which we do not.

Because of the uncertainties about the consequences of atmospheric contamination with asbestos, it is hardly possible to estimate the size of the health improvement from policy-induced reductions in exposures. It is possible to make some broad generalizations about the relationship between the amount of effort involved in reducing exposures and the scale of improvement in health to be expected. In general terms, it is relatively easy to control gross airborne contamination in industry. It is difficult to refute the suggestion that earlier acknowledgement that asbestos was a dangerous substance requiring serious attention and control would have made today's Commission unnecessary. Probably we would be at some conference arguing as to whether asbestos was a health hazard or not. It is more than likely that a similar pragmatic approach would be of most benefit in the public domain. Rapid attention to the more obvious sources of contamination as determined by visual inspection taking due account of the type of fibre is likely to be beneficial. Attempts to initiate action according to airborne sampling results are less likely to be based on genuinely useful criteria despite the attractions that numbers have to regulatory agencies.

It is evident that further information is required before reliable answers can be provided for the questions presented by the Commission. If asbestos is going to continue to be used, then careful long-term observations will be required. The most important observations relate to the measurement and characterization of airborne fibres. These must be taken so that individual exposures can be calculated. They must be taken so that the relation between standard counting techniques and total airborne fibre concentrations can be determined for each job category or work process.

In formulating regulations or proposals it should not be forgotten that the health benefit of any standard is likely to be related to the compliance with that standard on a day-to-day basis in the workplace. Ninety-five percent confidence that no worker will be exposed to greater than a certain fibre level may well have greater health benefits that 50% confidence in relation to some more rigid levels. This concept must take account of the reliability of individual measurements, of the frequency and strategy of sampling schedules, and of the credibility with which the limits really will be observed in industry. Defining very rigid standards which are ignored appears to be an exercise in futility for all concerned.

It is clear that the key questions posed by the Commission are, in large part, unanswerable, but we can start to define certain boundaries and to use these boundaries both to make decisions and to pose the correct research questions.

One key administrative mechanism has to be solved. It will be appreciated that asbestos is a complex group for substances. Even the species which I have mentioned vary considerably within themselves. Chrysotile, for example, varies greatly from one rock formation to another. It may be as unreasonable to assume that these different types of chrysotile have identical health effects as it is to assume that chrysotile mining in Quebec has the same health consequences as chrysotile insulation in Ontario. Long-term accumulation of knowledge by committed individuals will be required if solutions to these problems are to be forthcoming. Very considerable effort is required to find out more about exposure levels and to characterize the airborne fibres in very great detail. This is perhaps the most important area requiring sustained research effort.

APPENDIX D

ADDRESS BY:

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Kennedy School of Government
Harvard University

FRAMEWORKS FOR THE REGULATION OF HAZARDOUS SUBSTANCES IN JURISDICTIONS OUTSIDE CANADA

Prepared for the Royal Commission on Asbestos,
Second Public Meeting, December 12, 1980

Let me start off by stating my appreciation for the opportunity to come to your city. I am on leave from teaching at Harvard to work in a regulatory agency in Washington -- the Federal Trade Commission, which is the regulatory agency charged with consumer protection in the Unites States. And, although the thermometer may read a few degrees warmer in Washington this time of the year than it does in Toronto, let me tell you that just now the political atmosphere for somebody working in a federal regulatory agency in the wake of Governor Reagan's recent election, is absolutely frigid.

As you may know, Governor Reagan has not, in the past, been a particularly sympathetic observer of government regulatory programs in areas such as environmental protection or occupational safety and health. At one point, Mr. Reagan called for the total abolition of the Occupational Safety and Health Administration, the federal agency, part of the U.S. Department of Labor, in charge of the regulation of health conditions at the workplace -- although, during the campaign, he modified his position somewhat. In various remarks during the campaign, however, Mr. Reagan did suggest that trees were a more important source of air pollution than cars or factories -- and when Governor Reagan appeared at a college campus shortly after one of his statements to that effect, some students tied a banner between two large trees with the text, "Cut me down before I kill again."

So it is an anxious time to be a regulator. And, indeed, one of the themes I want to develop today is the growing political controversy over the government regulation of hazardous substances that has grown rapidly during the past decade. But I don't want to get ahead of my story.

My topic is the framework for the regulation of hazardous substances in selected jurisdictions outside Canada. I shall organize my talk as follows:

1. I will discuss in a bit of detail the regulation of occupational safety and health in the United States and Sweden. This is partly because this is the subject I know most about and partly because many of the patterns seen here are also seen in the rest of the regulatory framework for hazardous substances.

2. I will go on to discuss the regulatory framework for the rest of hazardous substances regulation, with special attention to the United States.

3. I will continue by discussing a bit how asbestos has fit into this pattern of regulation.

4. I will say a word about the situation in the United Kingdom.

5. I will conclude by discussing the recent controversies over government regulation in this area.

First, on occupational safety and health. There was a big upswing around the beginning of the 1970's in interest in occupational safety and health throughout most advanced Western countries. The Occupational Safety and Health Act in the United States was passed in 1970, which created an entirely new federal presence in this area. In Sweden, there was already an existing central government presence, but interest in occupational safety and health grew at the beginning of the 70's, and the Workers Protection Law of 1949 was amended and strengthened in 1974; and in 1976 a new Work Environment Law was passed. In Britain, also, there was a central government presence before the 1970's, but this presence was strengthened by the passage of the Health and Safety at Work Act in 1974.

The change was most dramatic in the United States, where occupational safety and health had traditionally been regulated only at the state level. An entirely new federal agency, the Occupational Safety and Health Administration (OSHA), was created in the labor department. This was part of the dramatic expansion of the role of government in American society that began around the mid-1960's. The first stage of this was the Great Society legislation that attempted to deal with problems of poverty in the United States. The second stage was the regulatory thrust of the late 1960's and early 1970's, centered around health, safety, and environmental issues. This represented a major change in American society, with many new demands being placed on business.

The political environment is very different in the United States and Sweden: Sweden has perhaps the strongest organized labor movement in the world, while the United States has one of the weakest; and Sweden was ruled for over forty years by a Social Democratic government. It is therefore interesting to see how the laws and implementing regulations in the area of occupational safety and health are both similar and different. My overall conclusions are that there are not insignificant differences between the statutes, but that, up to now, there has been a great similarity, in general, in regulatory decisions. (As will be noted later, asbestos in fact represents an exception to this generalization.)

Let us first compare the two statutes, the Occupational Safety and Health Act and the Work Environment Act:

1. Both statutes establish a vague but rather strict standard for determining the degree of protection that regulatory standards for hazardous substances shall aspire to. The Occupational Safety and Health Act talks about setting threshold limit values at a level that, to the extent feasible, no worker will suffer any ill-effects from his work experience, even though exposed to the chemical throughout an entire working life. The Work Environment Act in Sweden says that the work environment "shall be acceptable, considering the nature of the work involved and the social and technological progress in the society at large."

2. In both cases, the agencies are in reality given a great deal of discretion in determining levels of protection, given the relatively vague language in the statutes.

3. OSHA is allowed to set threshold limit values for hazardous chemicals, and to require medical exams for exposed workers, monitoring of hazardous substances in the workplace, and warning signs (especially used for carcinogens) in workplace areas. The statute does not authorize banning a chemical. In Sweden, bans are authorized "if especially significant from a safety viewpoint." The Swedish statute also authorizes the agency to prohibit the employment of certain workers if they are to be exposed to a chemical or an unsafe process to which they are especially susceptible. (There is no such statutory provision in the American Occupational Safety and Health Act, but OSHA has, in regulating substances such as lead where there may be a special damage to women workers, prescribed that workers removed from their jobs for occupational health-related reasons shall be entitled to "rate retention": that is, equal pay at their new job within the company.) The Swedish statute also authorizes the agency to require that employers get special permission from them before exposing workers to a given chemical. (A provision like this was suggested by the AFL-CIO in 1973 regarding OSHA standards for occupational exposure to carcinogens, but the agency rejected this approach at the time.) Finally, the Swedish statute specifically states that workers have a right to get information about hazardous substances they are exposed to. (In the United States, OSHA has proposed a regulation to this affect, although it has not been promulgated yet, and its fate under the new administration is uncertain.)

4. Both the Swedish statute and Swedish central collective bargaining agreements give great importance in occupational safety and health enforcement to local factory safety stewards and safety committees. These are required by statute and are also established by collective bargaining agreements nationally. They do not have authority to enforce the law directly, but the idea is that there will be local people "on guard" all the time about possible infractions of safety regulations. The employee side has a one-vote majority on safety committees. The Swedish statute also gives workers the right to stop work in an imminent hazard situation. This does not apply, however, to chronic exposures to hazardous chemicals; the situation is more one of an imminent safety hazard.

5. As regards sanctions for violating regulations, the United States has what I believe to be a uniquely "strict" enforcement system. The statute establishes a system whereby employers are expected to comply with regulations even before the inspector arrives at the workplace; the inspector thus metes out fines the first time a violation is uncovered ("first-instance sanction"). These fines are relatively small, however. The Swedish statute, by contrast, does not provide for first-instance sanctions; instead, a longer procedure is generally required before fines can be levied. In both statutes, there is provision for criminal penalties in certain instances. It should also be noted that the Swedes, as noted above, attach great importance to safety stewards and safety committees as, in effect, enforcers of the statute. There is also a relatively greater number of inspectors in Sweden than in the United States.

As for the regulations implementing the statutes in the two countries, their content has, up to now, been quite similar. Agency decision-makers have tended to choose more over less protective alternatives. They have also required use of engineering controls in dealing with hazards rather than personal protective equipment.

However, the process of developing regulations in the two countries has been very different. The American system is perhaps unique in the world regarding (1) the length of the procedure; (2) the amount of data gathered in the course of the procedure, particularly technical feasibility and cost data; (3) the degree of involvement of lawyers; and (4) the degree of involvement of courts.

I will give a brief and simplified description of the U.S. system. If it appears complicated and confusing, please realize that in reality it is even more complicated than I am indicating here. Please also note that the essential features of this system are duplicated throughout areas of the regulation of hazardous substances in the United States.

The first step is for the National Institute of Occupational Safety and Health to develop a "criteria document" that summarizes the toxicological and epidemiological evidence regarding the hazards of the substance in question and recommending a threshold limit value based on these hazards. OSHA next proposes an Advance Notice of Proposed Rulemaking (ANPR) in the Federal Register, the daily publication that presents information from administrative agencies. The ANPR includes the various alternatives the agency is considering, together with a "preliminary regulatory analysis" presenting the cost and benefits, in broad terms, of the various alternatives.

The next stage is for the agency to issue a "proposed rule" in the Federal Register. These are followed by lengthy public hearings, where just about anybody can testify and cross-examine witnesses. These hearings sometimes feature demonstrations, and typically feature lengthy examinations and cross examinations of witnesses, including witnesses from OSHA. Full

transcripts are kept of the entire hearing proceedings; the first round (of two rounds) of OSHA hearings on noise take up over twenty-five volumes.

Before the agency can issue a regulation, it must prepare an Environmental Impact Statement, detailing the effects of the proposed regulation on the external environment; a Regulatory Analysis, presenting the cost and benefits of the regulation; and under a new law just passed by Congress, a Small Business Impact Statement that considers the use of regulatory flexibility for different sizes of enterprise. When the regulation is finally issued, it must be accompanied by a "statement of reasons," that explains why the agency acted as it did and answers objections raised to its course of action in the course of the hearing proceedings.

All this procedure is then subject to court challenge, beginning with an Appeals Court and, potentially, going all the way up to the Supreme Court. Virtually every standard that OSHA has issued has been challenged in the courts, and some have made their way up to the Supreme Court. The OSHA's standard on benzene, for example, was thrown out by the Supreme Court last year. In Sweden, by contrast, it is infinitely easier to develop a regulation. Essentially, there are no statutory requirements for procedures that must be gone through to develop a regulation. What the agency basically does is to set up a committee with representatives of labor, management, and the agency; they sit down, and among themselves, develop a regulation. The parties do not confront each other, but sit around a table. I believe that the differences between the procedures in Sweden and the United States help explain the greater ability in Sweden to reach agreement on the contents of the regulation.

Next, I want to address how hazardous substances outside the workplace are regulated. In the United States, the key agency for the regulation of hazardous substances outside the workplace is the Environmental Protection Agency (EPA), set up in 1970 by an executive order of President Nixon, at a time of dramatic growth in environmental concern. The main statutes the agency implements which relate to hazardous substances include the following:

1. Clean Air Act of 1970: In addition to provisions regulating auto emissions and traditional air pollutants, there are also provisions for the regulation of so-called "hazardous air pollutants." The EPA can establish emissions standards and ambient air standards. The Clean Air Act is up for re-authorization in Congress next year, and there will be attempts to roll back some of its provisions.

2. Federal Water Pollution Control Act of 1972: Like the Clean Air Act, this Act allows the EPA to regulate both traditional pollutants and hazardous water pollutants. More attention has been paid in recent years to the regulation of hazardous water pollutants, as great progress has been made in the battle against traditional water pollutants.

3. Safe Drinking Water Act: This Act allows the EPA to set up threshold limit values for the presence of hazardous chemicals in drinking water.

4. Resource Conservation and Recovery Act of 1976: This Act deals with hazardous waste, a new issue that is beginning to get a great deal more attention in the light of incidences such as the Love Canal problem in New York.

5. Toxic Substances Control Act of 1976 (TOSCA): This is the most important act regarding the regulation of hazardous substances outside the workplace. It authorizes the EPA to set up a list of all chemicals in use in the United States; to require retrospective testing of chemicals already on the market when it is considered that not enough about their toxicity is known; to require companies to notify the EPA about intentions to market new chemicals and give the EPA the opportunity to require pre-market testing of these chemicals; and to adopt various restrictions on chemical production and/or use. These restrictions may include labelling requirements, exposure limits, production limits (a market-oriented approach that allows uses of the chemical in question, within production limits, to be determined by market forces), and product bans. (Note that OSHA does not have this last power in the workplace.)

Next, I would like to discuss how asbestos has fit into this general regulatory pattern in the United States and Sweden.

For OSHA, asbestos was one of the first substances OSHA regulated through its rulemaking procedures. In 1972, OSHA adopted a regulation requiring a phased-in reduction of the threshold limit value for workplace exposure to 2 fibers/cc, after allowing an initial exposure for the first few years of the regulation of 5 fibers/cc. This was the only OSHA standard that was appealed to the courts by the AFL-CIO and not by industry. Over the years, the concern that asbestos may have produced a major occupational health catastrophy -- highlighted by such things as the 1978 HEW report on occupational cancer -- has made asbestos the only substance that, having already gone through rulemaking, OSHA has re-opened for another rulemaking procedure. In 1975, OSHA proposed a lowering of the threshold limit value to .5 fibers/cc, and recently OSHA has announced an intention to propose a lowering to .1 fiber/cc. However, no Advanced Notice of Proposed Rulemaking has yet been issued on this, and it is unclear whether it will be issued before the end of the Carter Administration. The fate of such a proposal under the Reagan Administration is uncertain.

In Sweden, regulations adopted in 1964 were supplemented by new regulations developed in 1974. Shortly after these regulations were issued, there was a great deal of discussion in the press after discovery of some mesothelioma cases in Sweden. After this discussion, bans on asbestos use in spackles, glues, paints, and floor/wall coverings were introduced. Most controversially, the Swedish authorities banned the use of asbestos cement, except in asbestos cement pipe when used under certain controlled circumstances. The basic reason for this was the perceived difficulty in enforcing any occupational safety and health regulations for asbestos in construction workplaces where asbestos cement was used. The bans on asbestos are virtually the only bans that the Swedish occupational safety and health authorities have issued in conjunction with hazardous substances, which highlights the nature of the perceived problem. Asbestos is still allowed as a friction material in breaks, in personal protective equipment, and in some special purposes (such as

chloralkline electrolysis). The goal of the Swedish regulatory apparatus in this area is eventually to get rid of asbestos from working life.

In the United States, under the Clean Air Act, asbestos has been declared a hazardous air pollutant (along with such chemicals as cadmium and beryllium). Emissions limits have been set, but these are not particularly stringent (they merely require that there be "no visible emissions" of asbestos). No provisions regarding asbestos have been promulgated under the Federal Water Pollution Control Act, the Safe Drinking Water Act, or the Resource Conservation Recovery Act.

Under the Toxic Substances Control Act, asbestos has been a major area of concern for the EPA, along with PCB's and chloroflorocarbons. For schools, the EPA has proposed regulation requiring testing of schools for the presence of friable asbestos. The intention is, when this testing is completed, to propose another regulation for the clean-up of the schools. Again, action under the new administration is uncertain. Last year, Congress passed the Asbestos School Hazards Detection and Control Act, which authorizes grants to local school districts to cover 50% of the cost of asbestos clean-up in their schools.

Generally, the EPA has issued an Advanced Notice of Proposed Rulemaking under the Toxic Substances Control Act on asbestos. The agency says that it is considering bans of certain uses of asbestos and/or total limits on the amount of asbestos that can be used in the United States, with the market determining exact patterns of use. No rules have been proposed yet, and the fate is again unclear under the new administration.

The Consumer Product Safety Commission has investigated, together with the EPA, products containing asbestos and have indicated, in an Advanced Notice of Proposed Rulemaking, that they are considering bans on asbestos in various products.

In Sweden, a Product Control Act passed in 1973 establishes similar provisions to the Toxic Substances Control Act. It sets up a Product Control Council on whose board sit representatives for the Swedish equivalents to OSHA and the EPA. Given the actions undertaken regarding asbestos use in the workplace in Sweden, there has been less urgency on actions, such as bans, under the Product Control Act.

Next, I would like to say a word about the situation in the United Kingdom. The Health and Safety Work Act of 1974 united various of the disparate factory inspectorates that already existed (there were seven of them, part of five different government departments) into one agency. It also improved provisions for medical monitoring of workers. In general, however, less attention has been paid to occupational safety and health issues in the United Kingdom than in either Sweden or the United States. The statute talks aobut levels of protection for hazardous substances that are "reasonably practicable." The approach of the Health and Safety Executive regarding enforcement is similar to that in Sweden, except that there are not the same kinds of provisions for safety committees that the Swedes regard as an important feature of enforcement. In Britain's poor economic situation, the unions have not pushed as hard for strict occupational safety and health regulation as in the United States and Sweden.

Finally, I would like to discuss briefly the growth of opposition to government regulation of hazardous substances that has been apparent in the late 1970's, both in the United States and in other countries. Some of the factors in this opposition were particularly strong in the United States, given the suddenness of the new demands on business. Psychologically, many businessmen resented being interfered with. They also resented the increased amount of management time that dealing with regulatory issues came to take. Also, there is a relatively strong tradition of hostility to government in the United States. In both the United States and in other advanced industrial societies, the stagflation of the 1970's has provided an economic environment not favorable to regulation and allowed business to make the argument that regulation (or "over-regulation") has been an important cause of economic stagnation.

This growth of opposition to regulation has had several results. There have been far fewer new laws passed; in the United States, the Toxic Substances Control Act was the last major one, and it was passed only after a lengthy battle. There has been increasing use of economic analysis in regulation and increasing centralization of regulatory decision-making to the White House. The big question now is what will happen under the Reagan Administration. There have been suggestions for a one-year moratorium on new government regulations, suggestions for rollbacks of existing regulations, and a proposal that for every new regulation an agency adopts, regulations having equivalent costs to the newly promulgated regulation must be repealed.

Public attitudes have been much more nuanced. On the one hand, there is no doubt that, as a general proposition, the call to "get government off our backs" evokes wide resonance among many Americans. Added onto this has been the view, shared by most Americans, that the American economy is in bad shape, and that "the government" bears most of the blame.

But the evidence of public opinion polling also shows that this anti-government sentiment among the public does not -- and here is where the contrast to the attitudes of the business community are most striking -- extend to government health and safety regulation.

In a 1978 survey conducted by the Opinion Research Corporation, people were asked whether the costs added by regulation are worth it to protect workers' health and safety. Fifty-two percent said "yes" and only 12% said "no." On a question about whether the costs added by regulation are worth it to protect the environment, 50% said "yes," while 19% said "no."

And, just recently, when, within the space of a week or so, two major hotel fires in the United States claimed over one-hundred lives, the talk in the newspapers and on television was not of an overly protective and overly restrictive government, but of the need to strengthen fire safety codes in public buildings.

The fact is that, throughout the Western world, the special value attached to health as a representative for the special value associated with human life remains strong. I suspect that, in the short run, we will see greater and greater caution by the authorities in the regulation of hazardous substances. But, from a longer-run perspective, I am convinced that strict regulation of hazardous substances as risks to health corresponds to values and norms that have too important a role in our society to be shunted aside.

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